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CONTENTS

		PAGE
Ocular effects of vitamin-B–complex deficiency	*L. V. Johnson*	1233
Sensitivities of the color receptors	*J. Mandelbaum and E. U. Mintz*	1241
Neural mechanisms of visual localization	*S. A. Talbot and W. H. Marshall*	1255
Vitamin deficiency in Trinidad	*V. M. Métivier*	1265
Cephalic tetanus following ocular injury	*G. G. Jayme-Goyaz*	1281
Home training in orthoptics	*E. K. Stark*	1299
Monilial conjunctivitis	*R. F. Maddren*	1307
An ophthalmic lens transposition	*J. I. Pascal*	1308
The most useful and inexpensive ophthalmic knife	*C. A. Bahn*	1309

DEPARTMENTS

Society Proceedings	1311
Editorials	1320
Book Notices	1326
Abstracts	1328
News Items	1347

For complete table of contents see advertising page V

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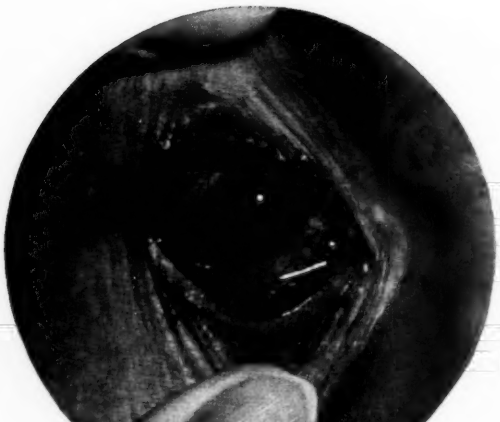

FIG. 7 (JOHNSON). MILD ROSACEA KERATITIS. NOTE THAT CAPILLARIES APPROACH BUT DO NOT INVADE THE INFILTRATE.

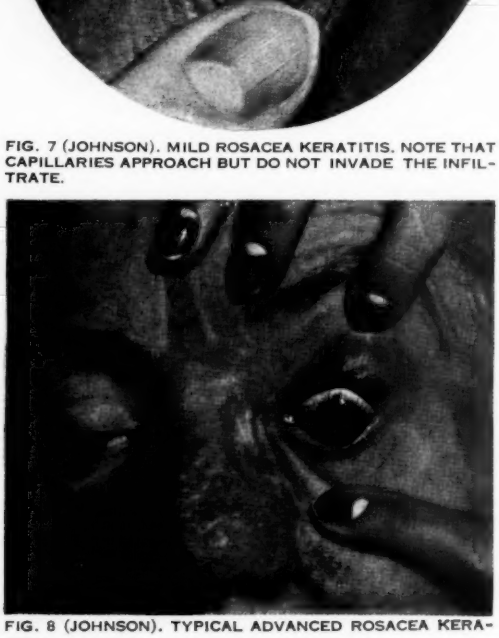

FIG. 8 (JOHNSON). TYPICAL ADVANCED ROSACEA KERATITIS.

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CLINICAL OCULAR CONDITIONS ASSOCIATED WITH VITAMIN-B–COMPLEX DEFICIENCIES*

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Several constituents of the vitamin-B–complex are concerned with oxidation-reduction reactions in cellular metabolism. For example: The phosphoric acid ester of *thiamin, co-carboxylase,* is known to be a co-enzyme in the oxidation of pyruvic acid in nerve tissue. It is believed that a lack of this vitamin results in the accumulation of pyruvic acid in the nerve, with resulting clinical manifestations such as polyneuritis and probably toxic (tobacco-alcohol) amblyopia.[1] While the role of alcohol (especially home-brewed wine and raisin brandy) and tobacco (especially cigars and pipe) are not definitely understood, the accelerated clinical decrease in the size of the caeco-central scotoma for red, green, and white test objects places thiamin chloride as an almost specific treatment for this condition. I have personally seen several cases of toxic amblyopia in which there was complete recovery within four weeks following adequate thiamin-chloride therapy, notwithstanding the continuance of previous habits as concerns alcohol and tobacco and diet. Less satisfactory response is observed in those patients who have experienced previous delirium tremens, and little or no response follows when the condition is of long standing and optic atrophy is present.

Nicotinic acid (or nicotinic acid amide, as preferably used clinically because of the absence of unpleasant symptoms of peripheral vasodilation) is known to be found in coenzyme II, which is undoubtedly concerned in oxidative systems. With lack of nicotinic acid, the clinical symptoms of pellagra are recognized. Ophthalmologists find particular interest in the following quotation from Sebrell:[2] "The very early writers on pellagra, such as Soler in 1791 and Rampoldi in 1885, refer to such eye symptoms as inflammation of the cornea, corneal ulcers and opacities, and among the early writers on pellagra in this country, Clark in 1909 reported pain in the eyes, conjunctivitis, failing vision, and iritis, and Whaley saw photophobia, mydriasis, and superficial inflammation of the cornea among other eye lesions in pellagrins. It seems not unlikely now that, in view of the close associations frequently seen between riboflavin deficiency and pellagra, many of these lesions may have been due to riboflavin deficiency. Last year Spies, Vilter, and Ashe again called attention to such lesions and stated that more than 70 percent of the patients in their nutrition clinic with frequent recurrences of pellagra, beriberi, or riboflavin deficiency also have visual disturbances."

Riboflavin (B_2) has long been known to be combined with phosphoric acid and

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Fig. 1 (Johnson). Vitamin-B-complex.

a protein to form Warburg's yellow enzyme. This yellow enzyme is present in all tissue cells, as a factor in tissue metabolism, and is believed to be particularly important in avascular tissues such as the cornea. Tissue oxidation does not occur by the simple proximity of gaseous oxygen and tissue metabolites. Rather, the body has found it necessary to utilize two systems of enzymes; one to activate the oxygen and the other to carry the hydrogen from the metabolites to the activated oxygen. Riboflavin appears to be concerned with the second of these enzyme systems.

After being activated by the dehydrogenase, the substrate itself becomes oxidized by giving up hydrogen to the coenzyme. The enzymatic nature of the yellow enzyme is indicated by the fact that it is reversibly reduced by the coenzyme, becoming reoxidized, without change, in the presence of molecular oxygen.[3] It is believed that if hemin substances are not present in a tissue (such as the avascular cornea), the oxidation

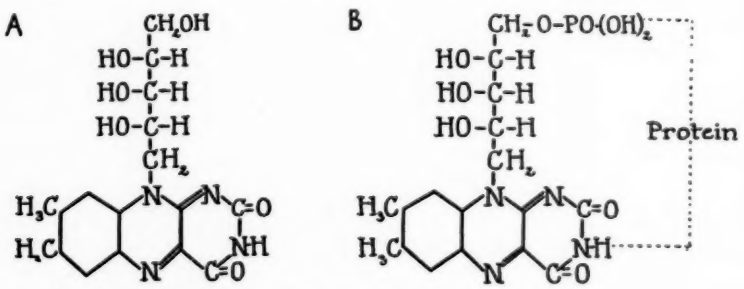

Fig. 2 (Johnson). A, riboflavin; B, Warburg's yellow enzyme.

within the cell is accomplished by Warburg's yellow enzyme. This fact is of particular interest in the investigation of the avascular cornea. A deficiency of riboflavin seriously impairs the oxidation system since riboflavin is the prosthetic group of Warburg's yellow enzyme. Pro-

substrate + dehydrogenase → activated substrate
activated substrate + coenzyme I → oxidized substrate + reduced coenzyme I
reduced coenzyme I + newer flavoproteins → activated reduced coenzyme I
activated reduced coenzyme I + hydrogen acceptor[12] → coenzyme I + reduced hydrogen acceptor

Fig. 3 (Johnson). Probable equations for mode of action of Warburg's yellow enzyme.

liferation of capillaries from the limbus would appear as an effort to combat localized anoxemia, by bringing hemin substances into closer tissue proximity. Regression of the corneal vascularization follows riboflavin sufficiency and the restoration of the yellow-enzyme system to its normal position in oxidation.

Rats fed a diet deficient in riboflavin develop a high incidence of vascularization of the cornea, and after riboflavin is added to the diet remission from the vascularization is prompt.[3]

Rats fed a diet deficient in riboflavin develop a variable incidence of cataract.[4] The earliest changes seen with the slit-lamp start in the nucleus, the change being rapid and accompanied by the formation of many vacuoles similar to those seen in dinitrophenol cataracts, except that the latter vacuoles early appear at the posterior and anterior cortex, while the rat cataracts are early nuclear. No specific changes in the human lens have yet been described as "ariboflavinosis opacities," and riboflavin appears to have little if any value in arresting the progress of human cataracts.

Choline may be essential for the production of acetylcholine, a parasympathetic stimulant of interest to ophthalmology because of its role in the physiological action of atropine, mecholyl, prostigmine, and eserine.[5, 6]

Pantothenic-acid deficiency in rats is associated with a high incidence of corneal ulcers, which tend to progress to perforation and endophthalmitis. Addition of pantothenic acid to the diet is followed by a definite tendency to healing.[7] If there is a human clinical ulcer associated with pantothenic-acid deficiency, it probably is the indolent, superficial loss of corneal epithelium and ulceration seen in elderly patients, which usually shows a central area of multiple discrete or conglomerate superficial staining, and of somewhat migratory character, tending almost daily to heal at one site and stain at another. Much remains unknown concerning the nature of action of pantothenic acid, and reference to a clinical entity is based only upon observations of clinical effectiveness fol-

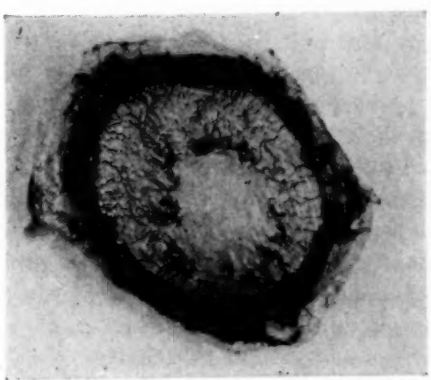

Fig. 4 (Johnson). Riboflavin deficiency of the rat cornea. The most characteristic features are the superficial nature of the capillaries and the abundance of Rouget cells.

lowing pantothenic-acid therapy. Much more definite experimental observations concern the observations on pantothenic-acid deficiency in the production of gray hair in black rats. It is observed that if these rats become sufficiently deficient, the hair becomes gray in color. The addition of pantothenic acid to the diet is followed by a change to a reddish-gray. If, however, *biotin*[8] is now given, the hair becomes a jet black, suggesting that the administration of pantothenic acid had thrown the rat into a deficiency of biotin. If, however, biotin is given first to the rat that is deficient in pantothenic acid, the hair becomes markedly whiter, and regains the black color again following a diet adequate in pantothenic acid. Still another vitamin, *para-aminobenzoic acid*[9] appears to be concerned with the color of hair. It is interesting that this vitamin also appears to inhibit the bactericidal effect of sulfanilamide on bacterial growth.[10]

It is already obvious that many almost synergistic therapeutic vitamin effects are present. In the field of medicine today, there is a relatively small group of investigators intent upon the minute study of isolated deficiencies and the results of specific treatment for reproducible symptoms. They suggest the course for the routine practice of the physician, who, as a practitioner, is concerned largely with the problem of securing the return to normal of all existing abnormalities. For practical purposes, they have provided for his use preparations of assayed vitamin products in such combinations that therapeutic amounts of all known, and probably many unknown factors are present. These commercial preparations contain a concentrated foundation from natural sources and are fortified, when necessary, with synthetic supplements to make them more effective therapeutically. Examples are: (1) extracts of rice bran, (2) unfractionated liver extract, (3) brewer's-yeast extract, each containing added amounts of the specifically indicated vitamins. The liver extract allows an intramuscular injection that is very potent. The use of such preparations insures a more balanced medication and prevents simultaneous untoward deficiencies, and thus should become a most desirable form of therapy, provided manufacturers maintain adequate concentration of the most indicated vitamins. The endless trade-naming of these preparations is now the only obstacle to their use for the average practitioner.

With this brief introduction to the vitamin-B complex, we will proceed to the condition of primary ophthalmological interest—so-called rosacea keratitis and the effect upon it of the administration of riboflavin. During the fall of 1937, Dr. Paul György granted me permission to follow, with regular slitlamp examinations, the progress of a series of rats maintained on a diet deficient in riboflavin. It was my desire to determine the type of biomicroscopic changes in the lens and to correlate them with any changes observed in human cataracts. As mentioned previously, no correlation was possible, but consistent vascularization of the cornea and the prompt remission, within a few days, following the administration of riboflavin were amazing. During the spring of 1938, when adequate amounts of riboflavin for clinical investigation were available, attempts were made to secure the regression of vascularization in a wide variety of corneal diseases. It was early apparent that the superficial vascularization accompanying trachoma, and the deeper vascularization associated with phlyctenular keratitis, sclerokeratitis, and interstitial keratitis were unaffected by any amounts of riboflavin. Neither was there any influence upon the inflammatory reaction in these conditions. Scarcely any

effect on this inflammatory reaction would be expected since riboflavin-deficient rats, with almost complete vascularization of the cornea, show no observable intraocular inflammation except hypopyon ulcer following secondary infection. It was immediately apparent that patients having so-called rosacea keratitis were promptly relieved following adequate administration of riboflavin, and that following the cessation of treatment, exacerbations were as promptly relieved.

I have continued the use of the term "so-called rosacea keratitis," for I have personally no clear definition of the entity. The condition is generally described as characterized by one or more small peripheral infiltrates (frequently scarcely discernible with the high-power objective of the slitlamp) between which and the limbus a varying number of small capillaries are present. These capillaries approach but do not invade the infiltrate (plate 2, fig. 7). The conjunctival vessels at the area become distended and usually are seen as an obvious wedge pointing to the area of the so-called rosacea ulcer. The "ulcer" is, however, in early development only an infiltrate that stains, and its healing is accompanied by the development of a small "facet," best seen by the reflected light with the flashlight held close to the cornea. Often, the patient with so-called rosacea keratitis presents several such small facets which remain from previous attacks. The differentiation between so-called small marginal and catarrhal ulcers is frequently most difficult. In my experience, the most helpful sign is the presence of the capillary loops in so-called rosacea keratitis, and absence of the bacterial conjunctivitis which is said to be the initiating factor in catarrhal ulcers. However, I have seen several rather persistent catarrhal ulcers clear completely in 24 hours following intravenous administration of riboflavin, and I question whether these too may not be sterile infiltrates at a site of local tissue anoxemia. Differential diagnosis of the vascularization, the other characteristic feature of rosacea keratitis, is even more difficult at times. Figure 8 (plate 2) shows so typical a cornea that there could be no question concerning terminology. The infiltrate is clearly discernible, as is the rather "wedge-shaped" or "triangular" area of vascularization upon which Doggart[11] placed so much emphasis. However, identical pathology, as frequently as not, occurs in patients without dermatologic acne rosacea. Given such a patient after the infiltrate has healed (particularly if it were small and no facet remained), the triangular characteristic of the superficial capillaries remains as the sole differential criterion. It is true that early in their development these vessels are capillary loops, exceedingly rich in Rouget cells, as judged by the marked variation in caliber of vessels, some capillaries being exceedingly small and others markedly engorged with cells. In long-standing disease, the vessels tend to lose this characteristic and appear to acquire something of a vessel wall; it then becomes more difficult to follow them as individual loops. Upon reaching this stage, they are not diminished by riboflavin and remain as permanent opacities.

Ophthalmologists agree that "rosacea keratitis" is a poor term, since the condition certainly is not caused by acne rosacea. Yet, the most severe cases are accompanied by acne rosacea, except in Negroes, who frequently have disabling rosacea keratitis without evidence of dermatologic acne rosacea. However, since most ophthalmologists understand these limitations in the terminology and apply them to the corneal entity under discussion, attempts to rename the condition would result only in further confusion.

The association of gastric achlorhydria with so-called rosacea keratitis, with or

without acne rosacea, has long been known and has caused many ophthalmologists to consider the condition one of the metabolic disorders. Also, it has long been known that the eye lesions, when recurrent, have tended to show the same exacerbations in women during the premenstrual days when metabolic demands in general are altered. Clinical experience has for years shown that remission from the attack is more prompt and exacerbations more infrequent following a diet that is low in carbohydrate and with the avoidance of coffee, tea, alcohol, and condiments. Since riboflavin is one of the enzymes capable of oxidizing carbohydrates, this reduced diet was quite possibly effective in sparing the riboflavin for the yellow-enzyme system. In December, 1939, in collaboration with Dr. R. E. Eckhardt, I released for publication original data collected during the previous two years concerning the observations on 36 patients with rosacea keratitis treated with riboflavin.[3] At that time, we frequently had observed that patients who early showed no HCl response to histamine, later showed almost normal levels. This has now become an expected circumstance, and it is our feeling that we may expect any number of patients, who were previously achlorhydric, to show a normal level following the intravenous administration of riboflavin. The same situation has been reported concerning thiamin chloride.[12]

Having made a diagnosis of so-called rosacea keratitis, it is our routine, at the University Hospitals of Cleveland, to administer an intravenous injection of 1 or 2 mg. daily of riboflavin and to advise the patient to take three teaspoonfuls of elixir of B complex daily until the lesion shows definite evidence of healing. Mild cases may require but two injections, but the oral therapy should continue over a week, and the diet should contain more than customary amounts of liver, milk, and eggs. When mild lesions have been recurrent, we advise that the oral B complex be continued in addition to the altered diet. For severe cases and those exhibiting considerable acne rosacea, we may continue the intravenous riboflavin, or intramuscular unfractionated liver extract (which does not have the vitamin factors precipitated out with alcohol as is done to concentrate the pernicious-anemia factor) for a more extended time, or until the histamine test shows the presence of gastric HCl. With this regime, the mild cases should be symptomless in 24 hours, and the infiltrate absent in 48 hours. The very severe cases, especially those with complete absence of HCl, may require a week to subside, and, as previously stated, we have not observed disappearance of the large vessels of long standing.

The richest natural food sources of riboflavin are liver, milk, and egg white. Generally, in nature, riboflavin occurs as riboflavin phosphate, and it is this compound that forms Warburg's yellow enzyme. Human breast milk contains riboflavin as the phosphoric acid ester, while cow's milk contains riboflavin in the free form. Commercial riboflavin is in the free form. Normally the intestine is able to phosphorylate riboflavin, prior to absorption. Riboflavin is rapidly destroyed *in vitro* when in an alkaline medium, and it is possible that the achlorhydria associated with rosacea keratitis further prevents the utilization of the vitamin in an adequate diet. Other speculative possibilities are: faulty conversion of riboflavin to Warburg's yellow enzyme; excessive excretion, necessitating unusually large dietary intake.

Clinical therapy would be facilitated if there were a simple, accurate laboratory determination of blood-riboflavin levels. The Snell-Strong[13] bacterial-growth method has been proposed for this, but for

blood determinations it is neither simple nor accurate. In this method, Lactobacillus casei ε is used as the organism. This bacillus does not synthesize its riboflavin and it does not grow unless riboflavin is present in the media. Growth is measured quantitatively by acid production. Application of the "Eckardt curve"[14] to blood-riboflavin determinations is advised, but it appears that blood levels are of no great practical value to the clinician. The accompanying chart (fig. 5) shows the results obtained when blood and urine riboflavin levels are taken after the intravenous injection of a reasonable amount of riboflavin. It is seen that the blood level of riboflavin shows no significant rise a half hour after the injection of riboflavin. On the other hand, it is apparent that the urinary excretion of riboflavin is markedly increased following the injection of

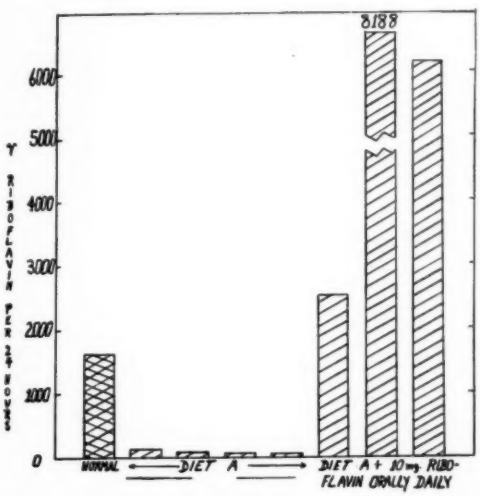

Fig. 5 (Johnson). Urinary excretion of riboflavin in micrograms per 24 hours.

3 mg. of riboflavin. The determination of riboflavin in the urine by this method is accurate, since the "unknown factor of Eckardt"[14] is not present in the urine. We have found that the 24-hour output of riboflavin in the urine is considerably reduced in patients who have rosacea keratitis (fig. 6). In two normal laboratory technicians eating a well-balanced diet, the 24-hour output of riboflavin in the urine was found to be over 2 mg. per day. This is considerably higher than the 500-800 micrograms reported by Strong *et al.*[15] However, even using their figure as

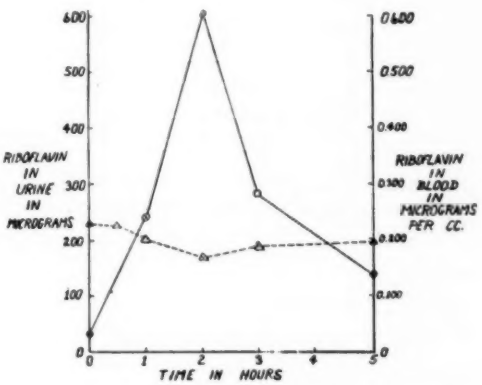

Fig. 6 (Johnson). Blood levels and urinary excretion of riboflavin following intravenous administration of 3 mg. riboflavin.

the normal daily output of riboflavin, the output of a patient with rosacea keratitis is seen to be considerably reduced. Following the daily administration of 10 mg. of riboflavin orally at the point indicated, the urinary output of riboflavin showed a considerable and significant increase to levels above the normal. It is possible that a riboflavin-saturation test may be devised as an index of riboflavin deficiency. Further work in this direction is contemplated in our laboratories.

Notwithstanding this evidence of rapid excretion of much of the riboflavin injected intravenously, very prompt relief from the infiltration and vascularization is observed clinically following the daily injection of 1 or 2 mg. Of particular interest is the observation that these rosacea patients, who have been repeatedly lacking in gastric secretion of HCl following histamine, will begin to show combined and free HCl after a few injec-

tions, and that oral riboflavin appears to become more effective with the presence of gastric HCl. It would be absurd to expect that the body could utilize or store any proportionate amounts of riboflavin when up to 20 or 25 mg. is suddenly injected into the blood stream, since these amounts probably represent the customary total intake of 10 days. Furthermore, it is to be recalled that riboflavin normally reaches the blood stream as the phosphoric acid ester and not the free form, and it is possible that such excessive amounts are simply excreted before phosphorylation can be effected by the blood.

Lakeside Hospital.

References

[1] Johnson, L. V. Alcohol-tobacco (toxic) amblyopia treated with thiamin chloride. Arch. of Ophth., 1939, v. 21, April, pp. 602-603.

[2] Sebrell, W. H. Nutritional diseases in the United States. Jour. Amer. Med. Assoc., 1940, v. 115, Sept. 7, p. 851.

[3] Johnson, L. V., and Eckardt, R. E. Rosacea keratitis and conditions with vascularization of cornea treated with riboflavin. Arch. of Ophth., 1940, v. 23, May, pp. 899-907.

[4] Eckardt, R. E., and Johnson, L. V. Nutritional cataract and relation of galactose to appearance of senile suture line in rats. Arch. of Ophth., 1939, v. 21, Feb., pp. 315-327.

[5] Clarke, S. T. Mecholyl and prostigmine in the treatment of glaucoma. Amer. Jour. Ophth., 1939, v. 22, March, pp. 249-257.

[6] Johnson, L. V. Newer concepts in the treatment of glaucoma. Ohio State Med. Jour., 1941, v. 37, Jan., pp. 36-37.

[7] György, P., and Eckardt, R. E. Further investigations on vitamin B_6 and related factors of the vitamin B_2 complex. Parts I and II. Biochem. Jour., 1940, v. 34, Sept., pp. 1143-1153.

[8] György, P., and Poling, C. E. Further experiments on nutritional achromotrichia in rats and mice. Proc. Soc. Exp. Biol. and Med., 1940, v. 45, Dec., pp. 773-776.

[9] Ansbacher, S. Para-aminobenzoic acid, a vitamin. Science, 1941, v. 93, Feb., pp. 164-165.

[10] Landy, M., and Wyeno, J. Neutralization (in vitro) of bacteriostatic activity of sulfonamides by p-aminobenzoic acid. Proc. Soc. Exp. Biol. and Med., 1941, v. 46, Jan., pp. 59-62.

[11] Doggart, J. H. Ocular rosacea. Trans. Ophth. Soc. United Kingdom, 1930, v. 50, p. 98.

[12] Williams, R. D., Mason, H. L., Wilder, R. M., and Smith, B. F. Observations on induced thiamine (vitamin B_1) deficiency in man. Arch. Int. Med., 1940, v. 66, Oct., pp. 785-799.

[13] Snell, E. E., and Strong, F. M. A microbiological assay for riboflavin. Ind. and Eng. Chem., Analyt. Ed., 1939, v. 11, June, pp. 346-350.

[14] Eckardt, R. E., György, P., and Johnson, L. V. Presence of a factor in blood which enhances the bacterial growth activity of riboflavin. Proc. Soc. Exp. Biol. and Med., 1941, v. 46, March, pp. 405-409.

[15] Strong, F. M., Feeney, R. E., Moore, B., and Parsons, H. T. The riboflavin content of blood and urine. Jour. Biol. Chem., 1941, v. 137, Jan., pp. 363-372.

THE SENSITIVITIES OF THE COLOR RECEPTORS AS MEASURED BY DARK ADAPTATION*

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New York

INTRODUCTION

A theory of color vision must primarily be concerned with a retinal mechanism capable of separating lights of different wave lengths into different neural-impulse patterns. The cerebral synthesis of color sensation is a second problem that is still obscure in the vast unknown of brain physiology; but it does not concern us here. This concept is not recognized in many of the published theories of color vision in which retinal and cerebral factors are often confused. Some theories of color vision—notably that of Hering—are based on the apparent "purity" of certain color sensations, such as white and yellow. That such "purity" is on a cerebral or psychological level and does not enter into the formulation of a retinal theory is readily shown by the binocular fusion of color images falling on corresponding retinal points.[6] Therefore, the premises underlying these theories make them inapplicable to the retina.

A theory of color vision must fulfill these general requirements:

1. It must be consistent with general physiological principles, such as the Müller doctrine of specific nerve energy. A sensory nerve, no matter how stimulated, can produce only its specific sensation; stimulation of a cone cell cannot produce more than one kind of sensation.

2. It must be consistent with a general theory of vision. Colored light does not differ fundamentally as a physical entity from white light; the effect of colored light on the retina cannot differ in any but a quantitative way from the effect of white light. Therefore a theory of color vision cannot be invented from whole cloth but must follow the same photochemical principles underlying all visual phenomena.

3. All the quantitative data concerning color vision and photopic vision in general must be consistent with the theory. These include color-mixture equations,[1, 10, 15] hue discrimination,[11] the photopic-visibility curve,[3] the saturation of spectral colors,[12] and other relevant data.

It is not the purpose of this paper to discuss all the theories of color vision that have been proposed; most of them can be discarded immediately because they do not meet the specifications outlined above. The Thomas Young Trireceptor Theory[17] offers a framework that permits the fulfillment of these conditions. According to this theory there are three types of cone cells, stimulation of each producing a pure sensation: red, green, or violet. White light stimulates these in a certain fixed ratio that may well be 1 : 1 : 1. Colored light stimulates them differentially according to their relative sensitivities to the specific color. Cerebral synthesis of the nerve impulses from all the cones stimulated results in the sensation of color. The original concept of vibrating fibers has given way to modern photochemical concepts. The three types of cone cells are now thought to have three photosensitive pigments with different spectral sensitivities. The pigments are bleached by light and regenerate spontaneously in the dark in a manner similar to visual purple.

* From the Laboratory of Biophysics, Columbia University. Read at the twelfth annual meeting of the Association for Research in Ophthalmology, at Cleveland, June 3, 1941.

"Excitation" curves describing the spectral sensitivities of the three color receptors have been constructed by many investigators, notable among whom were Koenig and Dieterici,[10] Abney,[1] and more recently Guild[4] and Wright.[15] All the formulations were derived from color-mixture equations. None of the formulations, however, could explain all the necessary data; furthermore there seemed to be no limit to the number of formulations possible. For these reasons the theory fell into some disrepute. All of the classical excitation curves have one feature in common: a wide distribution over the spectrum, with widely separated maxima. The curves computed by Weaver[13] from the classical data of Koenig and Dieterici and Abney (fig. 1) may be considered as typical of the many formulations; they fulfill the data of color mixture,[1, 10, 15] and the requirement for equal areas under the curves (to satisfy the theoretical consideration that for a white sensation all three receptors are probably stimulated equally); they are not consistent with the photopic-visibility curve.[3]

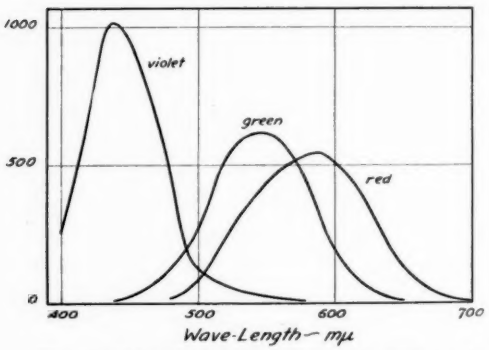

Fig. 1 (Mandelbaum and Mintz). Weaver's excitation curves, derived from the data of Koenig and Dieterici, and Abney. The ordinates are in units of intensity.

Hecht[5] recently introduced a set of sensitivity curves strikingly different from all earlier formulations. Instead of differing widely throughout the spectrum, his curves are close together; all have maxima in the yellow-green part of the spectrum near the region of maximum photopic visibility; the shapes of the curves vary but slightly from each other and from the photopic-visibility curve. The virtue of Hecht's formulation (fig. 2) is that for the first time much of the data relevant to color vision is included.

Obviously the commonly accepted sensitivity curves and the ones proposed by Hecht are preliminary attempts to discover the true shapes of the sensitivity distributions. Since these differ so strikingly in shape and position, it is important to make an early decision as to along which of the two paths further exploration should be conducted. With dark adaptation as a technique, we have performed experiments to test which of these concepts is correct. The principle underlying our work is that the brightness of the adapting or bleaching light determines the speed of the subsequent dark adaptation.[8] This is illustrated in figure 3, which shows the course of cone dark adaptation, as measured in a 1-degree central field with a green test light, following light adaptation to a blue-green light. As the brightness of the bleaching light is diminished (by the interposition of neutral filters)

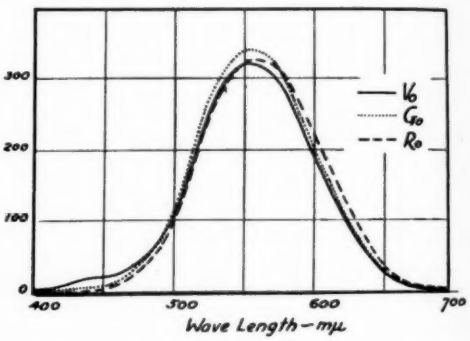

Fig. 2 (Mandelbaum and Mintz). Hecht's sensitivity curves. The ordinates are in units of intensity.

the speed of the subsequent dark-adaptation curve is increased.

If we light adapt with monochromatic light, the effect on each of the three color receptors will depend on its sensitivity to light of that wave length. If the violet receptor, for example, were not at all sensitive to extreme red light, as would follow from the classical curves, then measurements made with a violet test light following light adaptation to red light would fail to show any dark adaptation. Maximum sensitivity would be recorded immediately, for no bleaching of the violet receptor would have been effected. Dark-adaptation tests made with a red test light would be relatively slow; a green test light would indicate an intermediate speed. Similarly, following light adaptation to violet light, there would be no dark adaptation indicated if the test light were red; a violet test light would give a slow adaptation rate, and green would again give an intermediate speed. If, on the other hand, Hecht's theory were correct, all three receptors would be bleached by any spectral light. The differences in the dark-adaptation rates measured by the three test lights would be relatively small following light adaptation in any spectral zone. By comparing the speeds of the dark-adaptation curves obtained with the violet, green, and red test lights following light adaptation in different spectral regions, it is possible for us to estimate the relative sensitivities of the three receptor substances throughout the spectrum.

Wright[16] applied modified dark-adaptation measurements to this problem. In his experiments the right eye was light adapted, the left eye remaining dark adapted. Subsequently the right eye viewed a field of low illumination; the apparent color and brightness of this field were matched by means of three variable primary colored lights added together in a color-mixture apparatus and viewed by the left eye. As dark adaptation in the right eye progressed, the apparent brightness of the field it viewed increased, and increasing amounts of the three primary lights viewed by the left eye were required in order that a match be made by the two eyes. The rate of increase of each primary was plotted separately. Numerous objections can be made to this method, which is more closely related to color mixture than to dark adaptation. He measured changes in the eye already dark adapted rather than the course of dark adaptation in the right eye. The left eye, although considered dark adapted, actually became adapted to new levels of light intensity throughout the experiment. The difficulty of manipulating three independent lights in order to make not only color but also brightness matches with a constantly changing field is tremendous, particularly during the early stages of dark adaptation, upon which so much emphasis

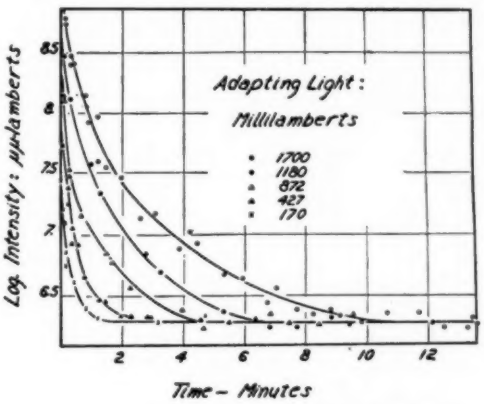

Fig. 3 (Mandelbaum and Mintz). Dark-adaptation curves illustrating the effect on the speed of dark adaptation produced by diminishing the intensity of the adapting light. The adapting light here was blue green; diminution in the intensity of the adapting light was achieved by the successive interposition of neutral filters of densities 0.16, 0.29, 0.60, and 1.00, respectively. Dark-adaptation measurements were made with a green test light. The points on the slowest curve represent three runs; the other curves are drawn through data from two runs.

is placed. Finally, the errors of binocular matching are introduced by this technique.

Apparatus and method

The observer sat in a cubicle in which were conveniently arranged the eyepiece and controls of the Hecht-Shlaer adaptometer[9] and the apparatus for light adaptation. A separate source for light adaptation was required in order to provide sufficiently high colored illuminations. We used a device similar to that employed by Hecht, Haig, and Chase.[8] A six-candle-power automobile-headlight lamp was the source of illumination. An image of the filament was formed on the observer's pupil by means of a double convex lens of focal length 4.5 cm. A filter holder placed in front of the lens accommodated "monochromatic" and neutral filters. This Maxwellian system produced a uniform retinal illumination over an area exceeding 30 degrees. The diameter of the observer's pupil always exceeded the size of the image of the light source in its own plane; for this reason it was not necessary to make corrections for pupil size.

The observer's right eye was first light adapted to an intensity of approximately 1,600 millilamberts for four minutes. Adapting intensities of this magnitude were best suited to our purpose. Lower bleaching intensities were followed by more rapid dark-adaptation curves, with a resulting loss in the precision of measurement; higher intensities gave unnecessarily slow dark-adaptation curves. In order to obtain violet light of the required intensity it was necessary to use a 50-candle-power lamp because of the great density of the violet filter. A heat-absorbing glass was used with the violet filter in this case, and the two were calibrated as a unit.

TABLE 1

Color filters used for light adaptation*

Filter Number	Color	Spectral Region in mμ	Central Wavelength in mμ	Density	Adapting Brightness in millilamberts
Corning 511	Violet	410–480	440	2.86	1520
Eastman 75	Blue-green	475–525	490	2.09	1700
Eastman 74	Green	520–550	535	1.55	1450
Eastman 73	Yellow	550–620	575	1.30	1780
Eastman 72	Orange	590–630	610	1.85	1550
Eastman 70	Red	650–700	675	2.15	1480
Eastman 88	Red	700–730	710	(Used only for test light)	

* The adapting brightnesses were obtained by combining color filters with neutral filters.

Table 1 lists the six color filters used for light adaptation and includes their spectral ranges and densities as measured with the Martens photometer by heterochromic matches (calibrated by J. M.). Since the transmissions of the colored filters differed and it was our aim to keep the adapting brightnesses nearly equal, it was necessary to interpose neutral filters in certain cases. The resulting light-adaptation brightnesses for each spectral region are given in column 6; the greatest difference is 0.09 log unit. A change of this magnitude in the adapting brightness will produce a perceptible difference in the speed of dark adaptation; however, since each spectral region is dealt with separately, this difference is of no consequence. The violet and green filters listed in the table also served for the violet and

green test lights; red filter no. 88 was used only for the red test light.

Following light adaptation, the first dark-adaptation reading was usually obtained within six seconds. After the first reading, more leisurely observations could be made. One or two additional readings were made during the first minute; subsequently one a minute sufficed. When a reading was made, the test light was exposed at intervals of six seconds. The duration of the test light was one-fifth second; it was 1 degree in diameter, exposed directly over the fixation point. The minimum intensity perceived as a circular field of light was designated as the threshold stimulus; the recorder noted the reading of the photometric wedge and the time of observation. The data were recorded by plotting the threshold (in log micromicrolamberts) against the time in the dark; the final threshold was established by at least two widely spaced readings at the same intensity. The data for identical conditions were easily reproducible; two runs were usually sufficient to define the shapes of the dark-adaptation curves.

The observer was occasionally troubled by after images. These occurred chiefly at the beginning of the run, and disappeared after a few seconds' rest. Following bleaching with violet and red lights, there were distortions in the apparent colors of the test lights during the first one or two readings; the test lights always appeared in their correct color after about a half minute of dark adaptation.

Results

Preliminary tests showed that light adaptation with white light was followed by similar adaptation rates for all three test lights. This is to be expected from the theory that the three receptors are equally sensitive to white light, and is consistent with the formulation of excitation curves with equal areas.

Dark adaptation was present when measured with all three test lights following light adaptation in all six spectral zones. This is inconsistent with the classical formulation of widely separated curves. According to this, the violet receptor, not at all sensitive to red light, would not have been affected by it, and, similarly, the red receptor would not have been affected by the violet light. In figure 4, typical data given by E. U. M. are reproduced; curves are drawn through the data obtained from separate runs. The dark-adaptation curves following light adaptation to blue-green light are given in figure 4A; here the shapes of the curves are most similar. In figure 4B the curves

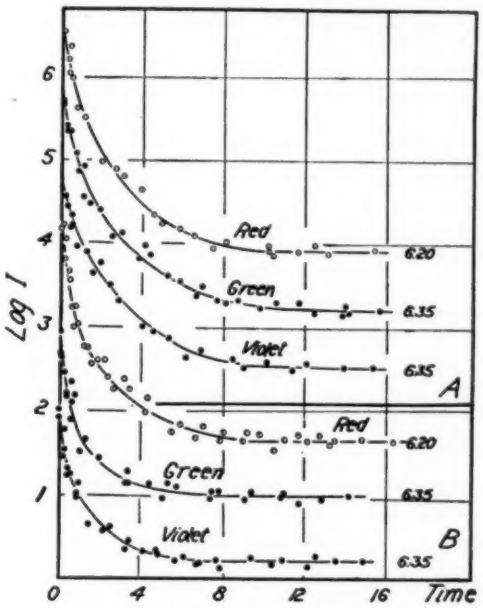

Fig. 4 (Mandelbaum and Mintz). Typical dark-adaptation curves. The curves have been displaced on the vertical axis; the actual threshold level (in log μμlamberts) is indicated for each curve. Figure 4A shows adaptation following bleaching with blue-green light; figure 4B shows adaptation following bleaching with red light.

following red light adaptation are shown, where the differences are greatest. The curves have been displaced vertically in the figure in order that they may be studied individually; the true values of the thresholds in log micromicrolamberts are indicated. E. U. M. gave a slightly lower threshold for red than for violet or green; two or three runs in each case. Single points are not given; the spread of our data is shown in figure 4. The curves for the red test light are correctly placed on the ordinate axis; the violet and green curves have been moved down about 0.15 log unit so that the thresholds coincide and the speeds of the curves may be compared

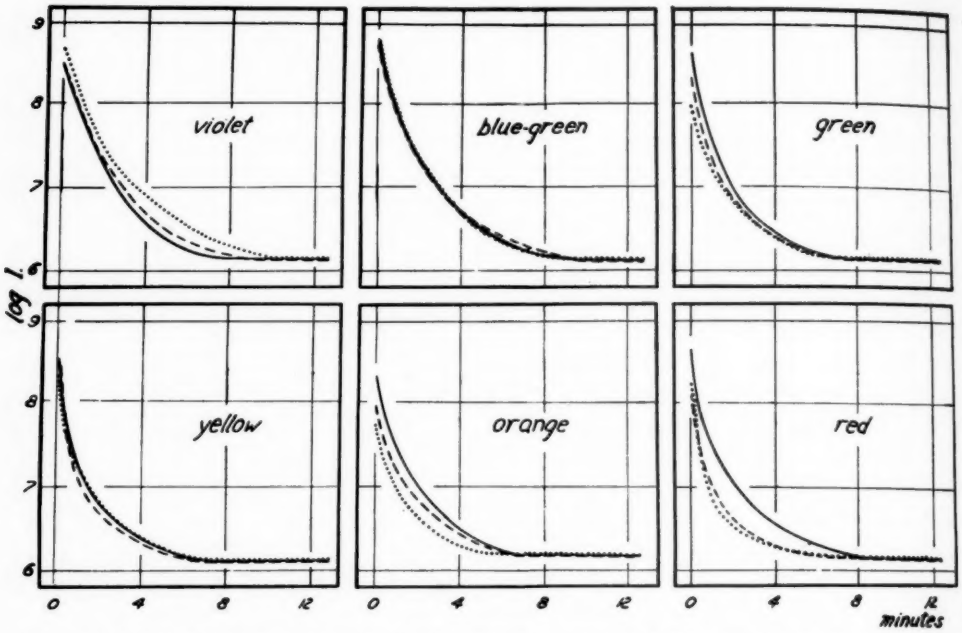

Fig. 5 (Mandelbaum and Mintz). Dark-adaptation curves following bleaching at different spectral regions, as indicated in each graph. The unbroken lines represent measurements with a red test light; the broken line the green test light, and the dotted line the violet test light. The curves represent averages drawn through the data from 2 or 3 runs. The curves are shifted vertically to make the violet and green thresholds coincide with the red.

this is probably due to a slight difference in the color sensitivity of her eye and that of J. M., who calibrated the filters. The difference is of no significance in our experiment, where we are concerned only with dark-adaptation rates and not with the final threshold levels achieved.

Figure 5 shows all the dark-adaptation curves obtained for the red, green, and violet test lights following light adaptation in the six spectral regions indicated in the figure. The curves are average curves drawn from data obtained from directly. The similarity in the shapes of the curves is at once more striking than the differences, which appear to be significant only after light adaptation near the ends of the spectrum.

A comparison of the speeds of the dark-adaptation curves obtained with the three test lights following light adaptation in any spectral region gives the order of magnitude of the differences in sensitivities of the three color receptors for that spectral region. If it were possible to separate the three receptors and to measure

the rate of regeneration of each separately, it would be possible to describe the differences in sensitivities exactly. However, the use of a violet, green, or red test light does not limit the retinal response to the corresponding receptor. This is not important if the spectral sensitivities of the three receptors are widely separated, as an examination of figure 1 reveals. Only a small error would be introduced if the retinal responses to the violet and red test lights were considered the responses of the violet and red receptors; in the case of green the error would be somewhat greater. But if the spectral sensitivities of the receptors are not widely separated we cannot neglect this error, and it would be fallacious to label the adaptation rates obtained with the three test lights the adaptation rates of the specific receptors. We shall refer to the retinal responses elicited by the red, green, and violet test lights as *r*, *g*, and *v*, respectively.

The speed of an adaptation curve may be measured by the time required to reach the final threshold. However, the slope of the curve is very slow as the threshold is approached, and a small error in intensity would lead to a large error in reading the time. The early parts of the curves are best avoided because the precision of measurement is not good while the adaptation is changing rapidly during the first minute. The distorted appearances of the test lights also occurred in this early region, and it is better to choose a less controversial region of the curve. It is therefore preferable to take as a measure of speed the time required to reach 0.50 and 0.25 log unit above the final threshold. Here the speed of the curves is sufficiently rapid, the precision of measurement good, and the separation between the curves adequate. In effect, we are measuring the time required to reach a specific stage in the regeneration of the photosensitive pigment. Two levels are chosen in order that the results may be averaged to add to their significance.

We examined our data in the region between 0.50 and 0.25 log unit above threshold in order to determine the precision of our measurements. From the deviations of 142 points in this region, the limits of accuracy of our determinations were found to be ±0.05 log unit. Differences in sensitivity smaller than this amount are without significance.

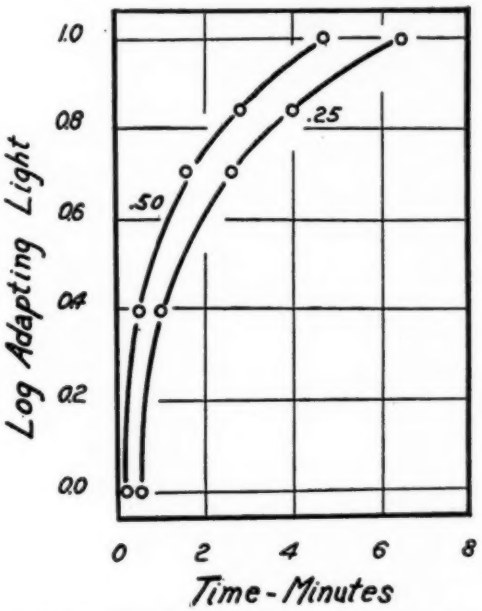

Fig. 6 (Mandelbaum and Mintz). The relation between the intensity of light adaptation and the speed of dark adaptation. The curves represent the time required to reach intensity levels 0.50 and 0.25 log units above the final cone threshold. The highest adapting intensity (1,700 millilamberts) is given a log value of 1.00. The data plotted here are from the curves shown in figure 3, and hold for the blue-green adapting light and the green test light.

When the intensity of the adapting light is cut down by means of the interposition of neutral filters, the speed of the subsequent dark adaptation is increased (fig. 3). The quantitative increase in speed is shown in figure 6, where the re-

lation between the adapting intensity (expressed logarithmically) and the time required to reach 0.50 and 0.25 log unit above threshold is plotted. The points in this figure are taken from the curves in figure 3, and pertain to dark adaptation measured with the green test light following light adaptation to the blue-green

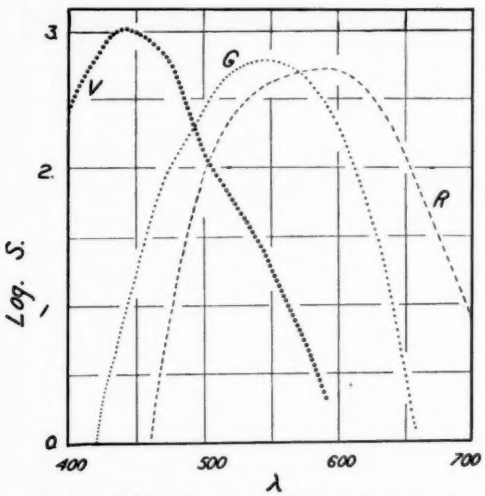

Fig. 7 (Mandelbaum and Mintz). Logarithmic plot of the classical excitation curves shown in figure 1.

light. The ordinates are arbitrary, the brightest adapting intensity (in this case 1,700 millilamberts) being given a logarithmic value of 1.00.

The quantitative differences in the sensitivities of v, g, and r toward the blue-green light can now be computed. Examination of the r curve (fig. 4A) reveals that it reached a level 0.50 log unit above its final threshold in 4.4 minutes; 0.25 log unit above threshold was attained in 5.9 minutes. From figure 6 it is evident that the g curve would have this speed if the intensity of the blue-green adapting light were diminished by only 0.02 log unit. The effective adapting intensity of the blue-green light for r is then only 0.02 log unit less than it is for g. Similarly, the v curve reached the two critical levels in 4.3 and 6.0 minutes, respectively; the interpolated values from figure 6 again give the effectiveness of the blue-green light for v to be 0.02 log unit less than for g. Conversely, the sensitivities of r and v toward the blue-green light may be considered as 0.02 log unit less than that of g. If, now, the arbitrary value of 1.00 log unit is assigned to the sensitivity of g toward the blue-green light, the sensitivities of r and v become equal to 0.98.

Since we are concerned only with relative sensitivities, it is more convenient to retain the logarithmic designation that our method yields. In order that we may compare our results with the original excitation curves, we plotted Weaver's curves

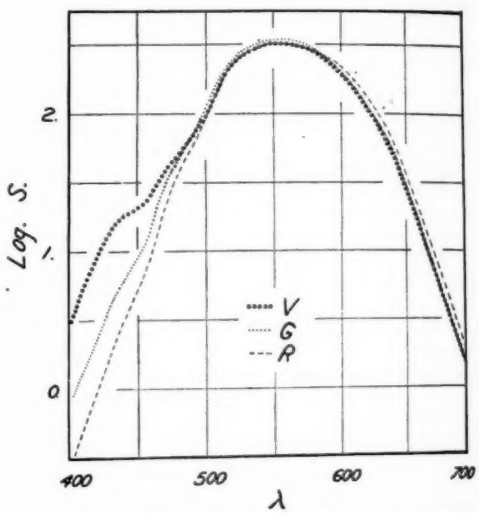

Fig. 8 (Mandelbaum and Mintz). Logarithmic plot of Hecht's sensitivity curves.

and Hecht's curves logarithmically; these are shown in figures 7 and 8, respectively. According to Weaver's curves, the sensitivity of the violet and green receptors should be about equal in the blue-green zone, and the sensitivity of the red receptor should be 0.70 log unit less. In order to obtain corroboration for this by our results, the red curve would

have to be much more rapid than we found it to be; as is apparent from the curves in figure 6, it should have reached our two critical levels in 20 and 60 seconds, respectively, instead of the 4.4 and 5.9 minutes it actually required. Therefore, even for the blue-green spectral region, where the classical sensitivity curves are closest, they are far too widely separated to be consistent with our data. Hecht's curves, on the other hand, are very close together in this spectral region, and are consistent with the negligible differences in sensitivity that we found.

Similar studies were made in each of the six spectral zones. In each case the speed of the slowest adaptation curve was increased by the interposition of neutral filters before the adapting light, and reference curves such as those for the blue-green light shown in figure 6 were drawn. The effective adapting intensities, and therefore the relative sensitivities of r, g, and v, were computed in each case. In the violet region, to give another example, the v adaptation curve was slowest. Reference curves were made by repeating light adaptation with violet light, cutting down the adapting intensity with neutral filters, and measuring each time dark adaptation with the violet test light. For the original maximum intensity, the v curve reached a level 0.50 log unit above its final threshold in 5.6 minutes; 0.25 log unit above threshold in 7.0 minutes. For the g curve the times were 3.9 and 4.9 minutes; for the r curve 3.6 and 4.7 minutes. From the reference curves it was calculated that if the effective bleaching intensity of the violet light towards v is given the logarithmic value of 1.00, the values for g and r become 0.85 and 0.81, respectively. Therefore, the relative sensi-

TABLE 2

DARK-ADAPTATION DATA*

Adapting Color	Dark-Adaptation Tests	Time to Reach (a) 0.50 Log Unit above Threshold	Time to Reach (b) 0.25 Log Unit above Threshold	Relative Sensitivities (a)	Relative Sensitivities (b)	Relative Sensitivities Average
Violet	v	5.6	7.0	1.00	1.00	1.00
	g	3.9	4.9	0.89	0.81	0.85
	r	3.6	4.7	0.83	0.79	0.81
Blue Green	v	4.3	6.0	0.97	0.99	0.98
	g	4.7	6.5	1.00	1.00	1.00
	r	4.4	5.9	0.98	0.97	0.98
Green	v	2.7	4.0	0.97	0.95	0.96
	g	2.8	4.1	0.98	0.96	0.97
	r	3.0	4.5	1.00	1.00	1.00
Yellow	v	2.7	4.0	1.00	1.00	1.00
	g	2.4	3.9	0.96	0.99	0.98
	r	2.6	4.0	0.99	1.00	1.00
Orange	v	1.6	2.5	0.80	0.75	0.78
	g	2.6	4.1	0.96	0.97	0.97
	r	2.9	4.3	1.00	1.00	1.00
Red	v	2.0	3.0	0.63	0.65	0.64
	g	1.8	3.0	0.58	0.65	0.62
	r	3.6	5.4	1.00	1.00	1.00

* Columns 3 and 4 list the times to reach points 0.50 and 0.25 log units of illumination above threshold. Columns 5 and 6 are computed from these data and are averaged in column 7.

tivities of v, g, and r toward the violet bleaching light are 1.00, 0.85, and 0.81. These figures are again consistent with Hecht's formulation. To obtain confirmation for Weaver's formulation, the order of magnitude of these differences would have to be very much greater, the difference between v and g being 2.00 log units, very close together, confirming Hecht's curves.

A summary of the data given by one of us (E. U. M.) is presented in table 2. The relative sensitivities of v, g, and r as determined by the time required to reach 0.50 and 0.25 log unit above threshold are given in columns 5 and 6;

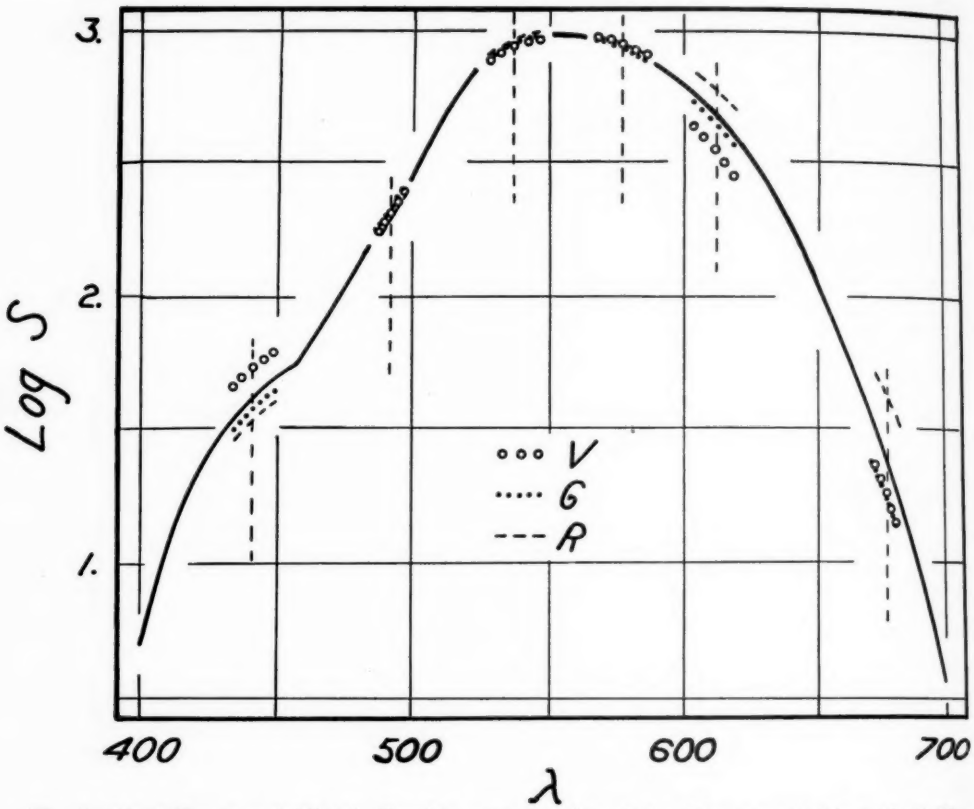

Fig. 9 (Mandelbaum and Mintz). Graphic representation of the data presented in table 2. The solid line is a logarithmic graph of the Gibson and Tyndall visibility data. The vertical broken lines indicate the spectral regions studied. The relative sensitivities of v, g, and r at these regions are indicated.

and the difference between g and r much greater than this.

Another critical region is the yellow. According to Hecht's curves, the three sensitivities are very close here, while in the classical formulation the red and green sensitivity should be equal, the violet lagging 2 log units behind. Again our results indicated that all three sensitivities are the averages of the sensitivity determinations are given in column 7. It will be seen that in the blue-green, green, and yellow spectral zones no significant differences in sensitivity were found by our method. This does not preclude actual differences, but indicates that the differences were less than our experimental error; namely, 0.10 log unit. Significant differences were

found only near the ends of the spectrum. In the violet region, at 440μ, *v* was most sensitive, *g* and *r* were almost 0.20 log unit less sensitive. At the red end of the spectrum at 675μ, *r* was most sensitive, *g* and *v* were almost 0.40 log unit less sensitive. A comparison of the data presented in table 2 with the logarithmically plotted excitation curves indicate immediately a fair fit with Hecht's formulation. Our results are incompatible with widely separated excitation curves.

We have attempted a graphical presentation of our data, based on the assumption that the sum of the sensitivities of *v*, *g*, and *r* throughout the spectrum should equal the photopic visibility curve. In figure 9 the solid line is the photopic visibility curve from the data of Gibson and Tyndall[3] plotted logarithmically. In each spectral region studied, the average of the three sensitivities was equated to the visibility ordinate. The difference of each sensitivity coefficient from the average was plotted above or below the visibility ordinate according to whether the sensitivity was greater or less than average. By drawing a curve through the *v* ordinates throughout the spectrum, we can indicate the spectral sensitivity of the *v* curve; in a similar manner we can draw the *g* and *r* curves throughout the spectrum. The ordinates of these curves are indicated in each spectral region; they are not drawn completely.

The dark-adaptation data given by J. M. were in essential agreement with the data of E.U.M. that have been presented. A complete analysis of his data was not undertaken.

Comment

The curves indicated in figure 9 represent the sensitivities of *v*, *g*, and *r*, which are functions of all the recipient cones, and not purely the violet, green, and red cones, respectively. The identification of these sensitivities with the sensitivities of the receptors themselves is valid in disproving the formulation of widely separated sensitivity curves, for here the error in making this identity is but slight, as has been explained. Moreover, the specific receptor was the dominant one stimulated in each instance; this is confirmed by the correct appearance of the color of the test light in each instance. Therefore it is permissible to consider the sensitivity curves, *v*, *g*, and *r*, which we have been able to draw throughout the spectrum, as a first approximation of the sensitivity curves of the color receptors. It is astonishing that these agree as well as they do with Hecht's curves, which have been carefully calculated from the data of color vision.

Hecht's curves were constructed in such a manner that they are consistent with the quantitative data of color vision relating to color mixture, hue discrimination, the saturation of spectral colors, the photopic-visibility curve, the neutral points of dichromats, and complementary colors. They were constructed, too, to have equal areas in order to conform with theoretical requirements. Finally, they are consistent with the data of cone dark adaptation, which we have just submitted. Therefore, they offer a more fertile field for future investigation than do the classical curves.

The requirements of a retinal theory of color vision as outlined in the introduction are now satisfied. The specific data of color vision have already been considered. The cerebral summation of primary impulses from discrete receptor cells is consistent with Müller's doctrine. The bleaching and regeneration of the receptor pigments is consistent with modern photochemical concepts of vision.[7] The three receptor pigments are in all probability varieties of the cone pigment visual violet or iodopsin, recently discovered by

Wald[14] and by Chase.[2] From a phylogenetic and from a photochemical point of view, the concept of closely overlapping curves is more probable than is that of the widely separated curves of classical formulation. Their differences in sensitivity may well be due to slight differences in their absorption spectra; for in the case of both visual purple and visual violet the spectral sensitivity is determined by the absorption spectrum. Such slight differences in spectral absorption could easily be accounted for by small molecular rearrangements, or the simple addition of a methyl or ethyl group. The presence of three closely related pigments and their development from a single precursor are more easily understood than would be the presence of three totally dissimilar substances with unrelated absorption spectra.

Conclusion

Experiments in cone dark adaptation, using colored adapting lights and test lights, gave results consistent with the formulation of closely overlapping sensitivity curves for the color receptors; the classical formulations of widely separated curves are inconsistent with our data.

The authors wish to express their indebtedness to Prof. Selig Hecht, who suggested this research, for his valuable help and criticism.

Bibliography

[1] Abney, W. de W. Researches in color vision. London, 1913.
[2] Chase, A. M. Photosensitive pigments from the retina of the frog. Science, 1938, v. 87, p. 238.
[3] Gibson, K. S., and Tyndall, E. P. T. The visibility of radiant energy. Sci. Papers Bureau of Standards, 1923, v. 19, p. 131.
[4] Guild, J. The colorimetric properties of the spectrum. Phil. Tran. Roy. Soc. London, 1931, v. 230A, p. 149.
[5] Hecht, S. The nature of the photoreceptor process. Handbook of general experimental psychology (Murchison). Worcester, Mass., Clark Univ. Press, 1934, pp. 781-821.
[6] ———. On the binocular fusion of colors and its relation to theories of color vision. Proc. Nat. Acad. Sci., 1928, v. 14, p. 237.
[7] ———. Rods, cones, and the chemical basis of vision. Physiol. Rev., 1937, v. 17, p. 239.
[8] Hecht, S., Haig, C., and Chase, A. M. The influence of light adaptation on the subsequent dark adaptation of the eye. Jour. Gen. Physiol., 1937, v. 20, p. 831.
[9] Hecht, S., and Shlaer, S. An adaptometer for measuring human dark adaptation. Jour. Opt. Soc. America, 1938, v. 28, p. 269.
[10] Koenig, A., and Dieterici, C. Die Grundempfindungen in normalen und anomale Farbensystemen und ihre Intensitätsverteilung im Spektrum. Zeit. f. Psychol. und Physiol. d. Sinnesorg., 1892, v. 4, p. 241.
[11] Laurens, H., and Hamilton, W. F. The sensibility of the eye to differences in wavelength. Amer. Jour. Physiol., 1923, v. 65, p. 547.
[12] Priest, I. G., and Brickwedde, F. G. The minimum perceptible colorimetric purity as a function of dominant wavelength with sunlight as neutral standard. Jour. Opt. Soc. America and R. S. I., 1926, v. 13, p. 306.
[13] Troland, L. T. Report of the Colorimetry Committee of the Optical Society of America. Jour. Opt. Soc. America, 1922, v. 6, p. 527.
[14] Wald, G. Photo-labile pigments of the chicken retina. Nature, 1937, v. 140, p. 545.
[15] Wright, W. D. A re-determination of the trichromatic coefficients of the spectral colours. Trans. Opt. Soc., 1928-29, v. 30, p. 141.
[16] ———. The measurement and analysis of colour adaptation phenomena. Proc. Roy. Soc., B, 1934, v. 115, p. 49.
[17] Young, T. On the theory of light and colors. Lectures in Natural Philosophy, London, 1807, v. 2, p. 613.

Discussion

Dr. Samuel A. Talbot (Baltimore): Would the essayist discuss Wright's conclusion from mixture technique in describing purely chromatic recovery after monochrome adaptation, taking into account the nonlogarithmic law which describes pure chromatic recovery as apart from recovery of brightness-threshold? Wright's conclusion was the opposite of this essay.

Dr. Mandelbaum:* From his data Wright concludes that photochemical regeneration occurs at a uniform rate for each receptor regardless of the adapting intensity (within limits); that there is a "specific effect on each receptor as well as a general depression of sensitivity"; and that the data are consistent with his excitation curves, modified slightly. However, at levels of light adaptation above 1,500 photons, "abnormal" effects are produced and the visual processes break down.

Analysis of his data shows that his points, although they appear to lie on straight lines, can nevertheless be described by curves that fit the data as well or better. His extrapolated values for time equals zero lose their significance. It is not surprising that his data should give what appear to be almost straight-line functions; this is the inverse of the almost hyperbolic curve, which is the customary manner of describing dark adaptation. The sum of the three single recovery curves must equal the total brightness threshold curve (for a brightness match as well as a color match was made). The shape of this function and its dependence on the previous light adaptation have been well established. This is inconsistent with the straight-line, uniform recovery rates which he describes.

The "abnormal" responses to which he refers, occurring with adapting brightnesses higher than the moderate level of 1,500 photons, are not shown in any data of the various retinal functions which have been studied at photopic levels. He observed that following light adaptation in any spectral region, all three retinal processes are markedly affected. This confirms our results. However, he misses entirely the significance of this finding, postulating instead a vague general and specific effect.

The slight transformations he makes on his previous excitation curves are based on the assumption that the pure sensations can be matched by spectral colors. This is in conflict with the finding that all spectral colors affect markedly the three responses. His new excitation curves represent again the old color-mixture data in a slightly modified form, with nothing significant added.

Dr. Talbot: Would your adapting lights have enough spectral width to excite widely separated curves?

Dr. Mandelbaum: Our adapting lights were not monochromatic. We used the Eastman Kodak so-called monochromatic filter series. These gave a considerable range of spectral transmission with maxima that were fairly well defined. The data for the spectral transmission of our filters were given in table 1.

Dr. Snell (Rochester, New York): Dr. Mandelbaum, would your sensitivities curves be modified in any way by the pigment in the personality; that is, the difference between the fair and those of dark complexion?

Dr. Mandelbaum: Those differences would be reflected in color-mixture equations and in the photopic-visibility curve, if they were to appear. As far as I know,

*The brief discussion of Wright's work given in the text of the paper was not included in the oral presentation, but was presented at this point, preceding the elaboration which follows.

no correlation has been found between the pigmentation of the skin and the hair, and the photopic-visibility curve or color-mixture equations.

DR. SNELL: There was a statement made by some of our Army investigators to the effect that the colored race has a greater degree of sensitivity in the dark—a more efficient adaptability—than the white race, and they hoped perhaps to employ the colored race as automobile drivers at night.

DR. MANDELBAUM: I have studied dark adaptation on many patients with dark complexions but unfortunately never on a colored person. If their light threshold is lower, their visual efficiency under poor illumination would undoubtedly be higher.

DR. G. L. WALLS: (Detroit): What was your reason for using stimuli equated in objective intensity rather than in subjective brightness for light adaptation?

DR. MANDELBAUM: The colored filters were calibrated on the Martens Photometer by means of heterochromic photometry; the filter factors obtained were used to equate the adapting brightnesses. Since subjective factors are concerned in heterochromic photometry, our adapting brightness must be considered to be matched subjectively rather than in objective intensity.

PHYSIOLOGICAL STUDIES ON NEURAL MECHANISMS OF VISUAL LOCALIZATION AND DISCRIMINATION*

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Baltimore, Maryland

The electrical method of determining cortical localization of response to physiological stimuli,[1] now makes possible a comparison of the retinal and cortical mosaics.

TECHNIQUE

We have mapped the cortical field of cat and Rhesus monkey, under chloralose and barbiturate anesthesia, by the following system (fig. 1): A moist thread on the exposed pia, or an insulated needle reaching to an inner striate surface, leads to an amplifier the action potential evoked by light in the visual field. An electrical light valve of large aperture was devised to make sufficient energy available over a large test field, within the utilization time of the eye. If the visual pattern is narrowed down to a band and then to a small square, a position can be found in the field where a movement of a degree or less will reduce the response at the cortical point. This response is observed periodically on a cathode-ray tube, and the stimulus moved by manipulating crossed slits, until the primary response shows maximum amplitude and minimum latency for the least stimulus area.

Under this condition of minimal stimulation, the stray retinal excitation is subthreshold, even with the highest brightness (10 ml.) used. With increase of angle from gaze, the integrating action of the retina requires minute stimuli for accurate localization in the field; but reduced sensitivity there demands more brightness. In both cat and monkey the retinal locus of greatest cortical sensitivity in darkness is several degrees below the central area. The anesthetic seems to reduce sensitivity, especially in the monkey, so much that 6 photon-sec. of energy is required from a spot covering 3 sq. deg. in central vision. Dark adaptation lowers the photic threshold only about 1.5 log units in peripheral vision; apparently the mechanism is depressed. It seems to be partly restored by injection of vitamin A.

The animal is mounted in a Horsley-Clark holder designed for wide visual field (fig. 2). With this instrument, a needle tip may be located in buried cortex conveniently for subsequent histological check. As many as four loci appear at different levels in the cat's brain. Tilting the monkey's head as shown (fig. 1) reduces occipital herniation; intraperitoneal glucose improves the condition of the preparation during the 12 or 15 hours with open skull. The cornea remains functional if irrigated, but maintains better images when protected by a contact glass.

The steadiness and orientation of gaze constitute major problems. We have tied the conjunctiva near the corneal limbus to a wire ring (fig. 2), supported in a clamp which affords various rotations about the center of the eye. Even slight movements would spoil measurements of high precision taken over several hours. The gaze and tilt of the eye are determined and adjusted with the help of an ophthalmoscope (fig. 3) suitably mounted. With this instrument, the blind spot and central area are optically projected to

* From the Laboratory of Physiologic Optics of the Wilmer Ophthalmological Institute, Johns Hopkins Hospital and Medical School. This work was supported in part by the John and Mary Markel Foundation. Presented at the twelfth annual meeting for the Association for Research in Ophthalmology, at Cleveland, June 3, 1941.

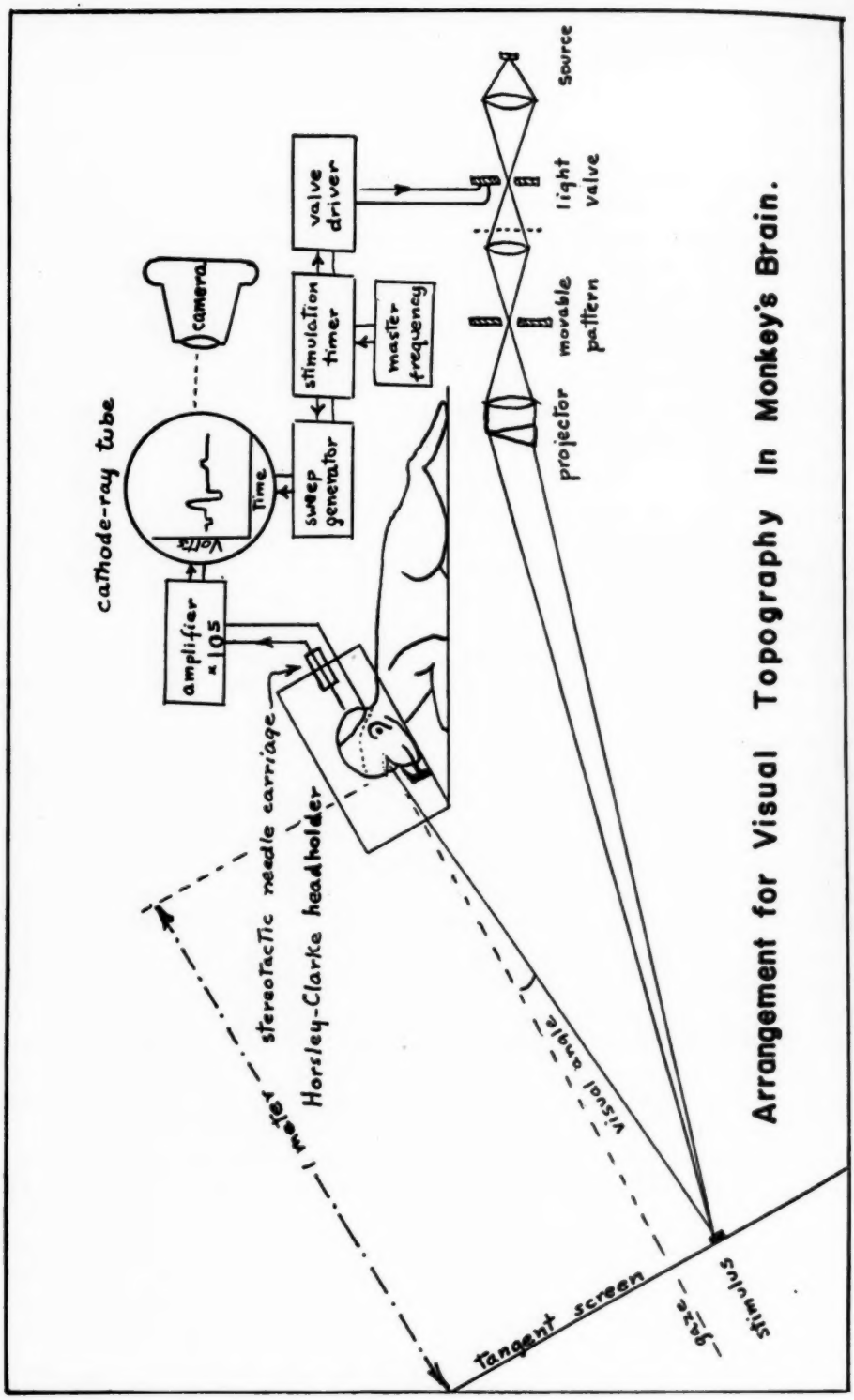


Fig. 1 (Talbot and Marshall). Experimental arrangement, showing posture of animal, stimulating and observing apparatus.

points on the test field that serve as reference for stimuli. This instrument also maps the tapetum and the orbital limits of the field.

Results

The resulting cortical maps are more detailed and differ somewhat from the anatomical pictures of Poliak[2] and Minkowski.[3] In both monkey and cat (fig. 4) each hemi-cortex projects to the contralateral half-fields of both eyes, the line of division splitting the fixation point. Since these fields touch, but within 0.5 degree do not overlap in any animal so far, we find no biprojection of the macula. The vertical meridian in the cat[4] extends for 25 mm. along the dorsal marginal and

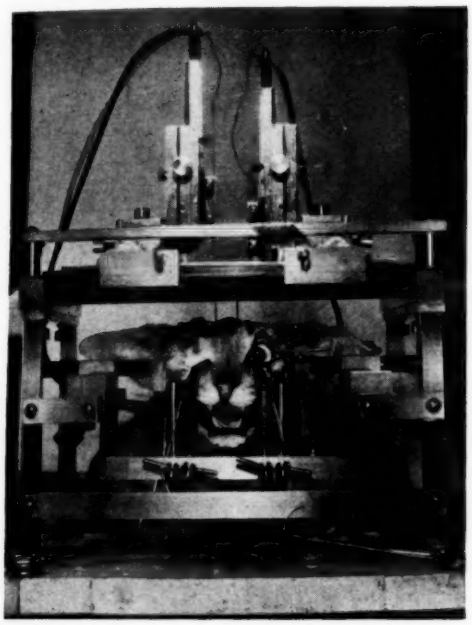


Fig. 2 (Talbot and Marshall). Horsley-Clark holder with free visual field and two needle carriages.

postmarginal gyri, reaching 40 degrees below, and 20 degrees above, the gaze at 0. Horizontal vision can be followed down the medial wall and lateral on the roof of the splenial sulcus to more than 50 degrees from gaze. The temporal crescent apparently has very small representation.

The Rhesus operculum is more interesting in regard to visual function, and

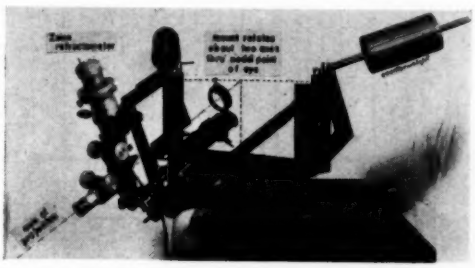


Fig. 3 (Talbot and Marshall). Instrument for determining gaze and tilt of eye.

has been mapped in more detail (fig. 5). The vertical meridian courses from 6 degrees below gaze at the top, around the striate, and disappears into the inferior sulcus about 4 degrees above gaze. The half-fovea is projected to an area 6 mm. in radius, situated about 5 mm. below the tip of sulcus lunatus near the ear, and lateral to the depression suggested by Poliak as a cortical fovea. Eight degrees of the horizontal meridian usually appear on the exposed cortex. The medial half of this exposed meridian lies over the line where the internal calcarine meets the inner opercular face horizontally. On this inner line the horizontal meridian then goes lateral 15 mm. to about 14 degrees, before turning rostrally.

The laterally convex arrangement of contours shows that a sagittal lesion would produce crescentic scotomas. The spacing of contours is inversely proportional to discrimination on the cortex. In 36 cats, the cortical discrimination averages 5 degrees per millimeter of cortex at 30 degrees below gaze, but 1 deg./mm. centrally. In six monkeys, the discrimination averages 18 minutes of visual angle per millimeter of cortex in the 5-degree periphery, but increases to 2 min./mm. cen-

Fig. 1 (Talbot and Marshall). Experimental arrangement, showing posture of animal, stimulating and observing apparatus.

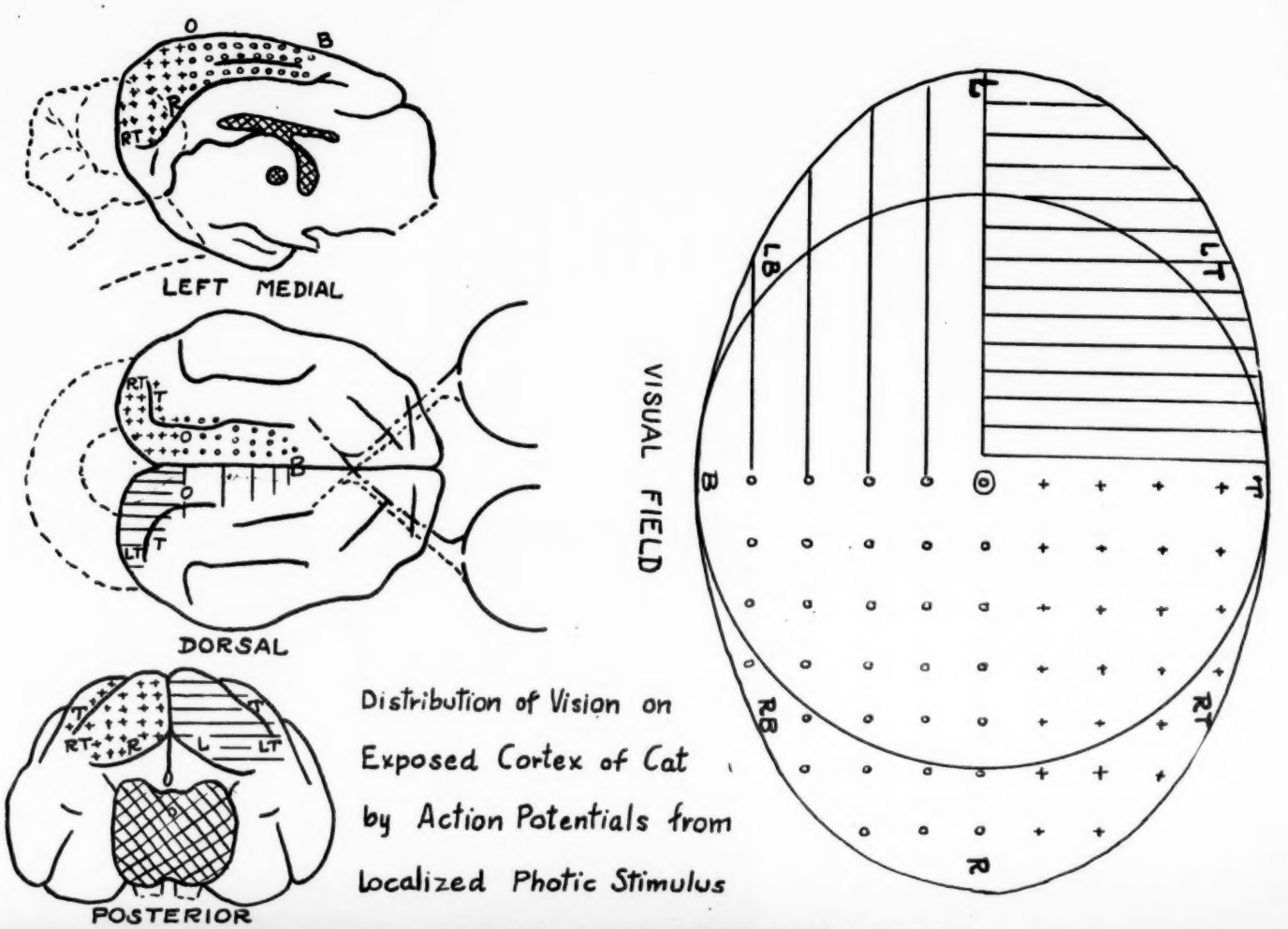


Fig. 4 (Talbot and Marshall). Projection on field of cat's cortex.

Fig. 4 (Talbot and Marshall). Projection on field of cat's cortex.

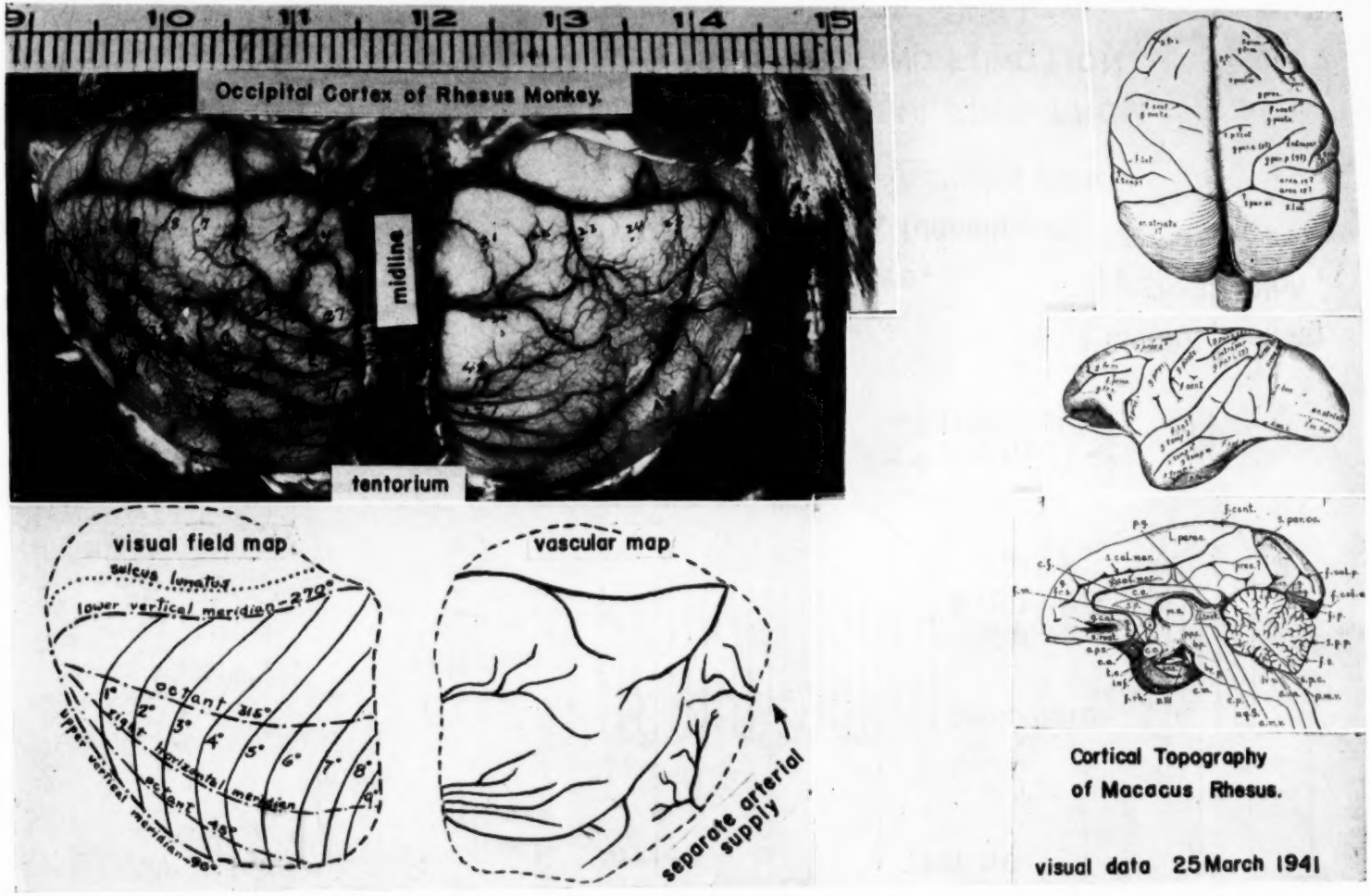
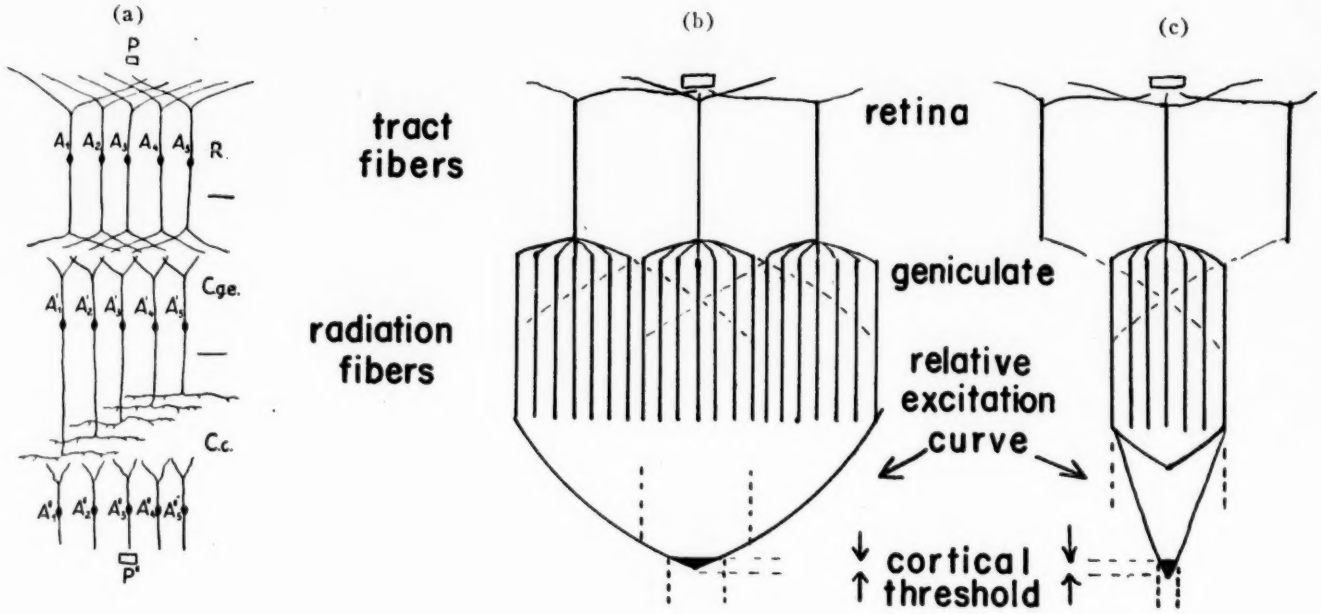


Fig. 5 (Talbot and Marshall). Projection of field on monkey's operculum.



LABILE LOCALIZATION RESULTING FROM GENICULATE STRUCTURE AND FUNCTION.

Fig. 6 (Talbot and Marshall). a. Channeling by reciprocal overlap (Lorente de Nó). b. Peaking by overlap at a multiplying synapse. c. Peaking by facilitation in an intensity pattern.

trally. Such a cortical spread would justify more refined experiments near gaze, using very small stimuli to investigate the limit of cortical localization in terms of the overlap of unit paths.

Discussion

These results show that a 1-min. circle at the fovea (ca. 0.005 mm.) is magnified 10,000 times in area to cover a 0.5-mm. circle at the cortex. The immense multiplication of primary path which this connotes, carries several important consequences for those types of acuity which exceed retinal structure. The integrations and averaging involved in vernier or stereoscopic estimates need no longer be ascribed mainly to associative or other higher activity.

A one-to-one relationship in ascending neurons (fig. 6a) would cause impulses to remain channeled in proportion to the reciprocal overlap, as Lorente[5] showed some years ago. But at a geniculate layer (possibly for each color,[6]) for each optic fiber from each eye there are many[7] radiation fibers interconnected by extensive reciprocal overlap[8] (fig. 6b). Thus the retinal mosaic supplies a much finer-grained cortical mosaic, and the distribution of excitation is highly peaked by the overlap. Depending on cortical threshold, it may be even narrower than the initial excitation at the retina. To this must be added the effect of facilitation of successive stimuli at the geniculate[9, 10] and cortex,[11] which we have recently demonstrated experimentally. The distribution of intensity (that is, impulse frequency) across the geniculate pattern causes the central portion of the image to be further augmented in strength (fig. 6c), so that the cortical image may well be 1/10 of a cone unit, as has been observed.[12, 13] It can also be shown that this effect must raise acuity with length of the test line, as observed. Similarly, the facilitation we have found at the cortex between converging fibers of the two eyes[11] helps account for binocular increase of acuity.

Moreover, the combination of multiplied paths rich in reciprocal overlap, with depth projection and binocular facilitation, affords a more detailed mechanism for binocular fusion (fig. 7) than heretofore proposed. Oppositely oriented disparate images in the two geniculate layers converge to common neurons at the cortex, where the physiological fluctuation permits facilitation to fuse correspondinng parts of the pattern into a posture related to the original object. More adequate discussion cannot be given in the limits of this paper than to mention the relation of fusion to the neural system we are describing.

Finally we must reëmphasize the importance of such discontinuity in visual functioning. The play over the cortex of waves of excitability associated with spontaneous Berger activity is continuously modulating the refinement of imagery there by its effect on threshold. At the other end, the velocity and frequency of physiological nystagmus[14] utilizes what we have reported[11] on the effect of cyclic stimulation. The oscillation of retinal pattern across the receptors is such as to augment facilitation and so sharpen the neural image. Consequently no *static* picture of structure and function can account for the known facts of hyperretinal discrimination or contrast.

Thus within the framework of striate localization described at first there exists the interconnection of structure and lability of function needed for the finer patterns perceived. A one-to-one projection of retina on cortex exists functionally only in a dynamic sense.

Fig. 6 (Talbot and Marshall). a, Channeling by reciprocal overlap (Lorente de Nó). b, Peaking by overlap at a multiplying synapse. c, Peaking by facilitation in an intensity pattern.

GENICULATE STRUCTURE AND FUNCTION.

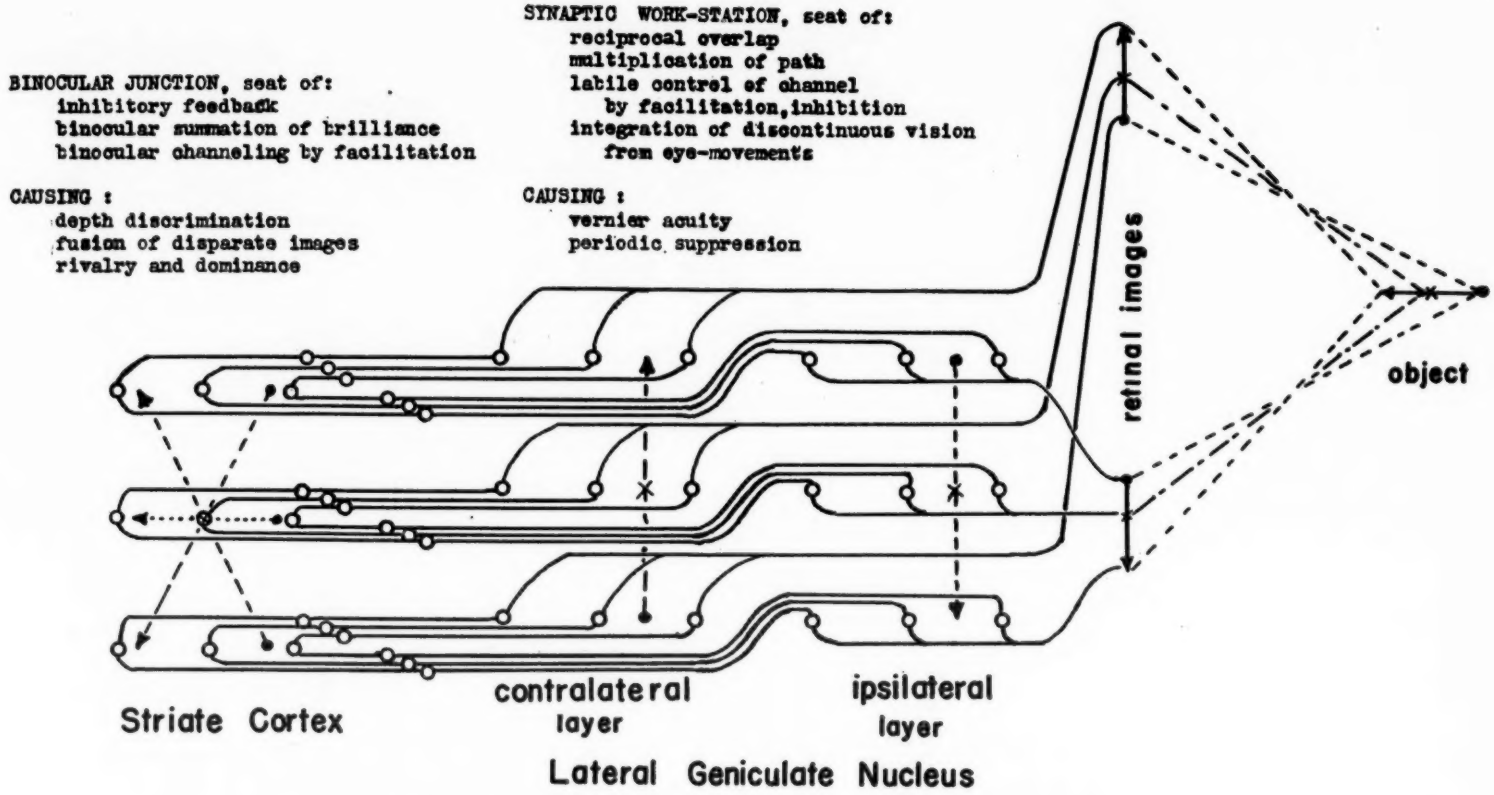


Fig. 7 (Talbot and Marshall). Proposed scheme for binocular fusion, using multiplied path, facilitation, and fluctuation mechanisms.

References

[1] Marshall, W. H., Woolsey, C. N., and Bard, Philip. Observations on cortical somatic sensory mechanisms of cat and monkey. Jour. Neurophysiol., 1941, v. 4, pp. 1-43.

[2] Poliak, S. Main afferent fiber systems of the cerebral cortex in primates. Univ. of California Publications in anatomy, 1932, v. 2, pp. 107-207.

[3] Minkowski, M. Experimentelle Untersuchungen über die Beziehungen der Grosshirninde. Arb. a. d. Hirnanat. Inst., Zürich, 1913, v. 7, p. 259.

[4] Talbot, S. A. Arrangement of visual field on cat's cortex. Proc. Amer. Physiol. Soc., 1940, pp. 181-182.

[5] Lorente de Nó, R. Studies on the structure of the cerebral cortex. Jour. Psychol. u. Neurol., 1934, v. 46, pp. 133.

[6] Clark, W. E. L. Anatomical basis of colour vision. Nature, 1940, v. 146, pp. 558-559.

[7] Glees, P., and Clark, W. E. L. Termination of optic tract fibers in lateral geniculate of the monkey. Jour. Anat., London, 1941, v. 75, p. 295.

[8] O'Leary, J. L. A structural analysis of the lateral geniculate nucleus of the cat. Jour. Comp. Anat., 1940, v. 73, pp. 405-430.

[9] Bishop, G. H., and O'Leary, J. L. Electrical activity of the lateral geniculate of cats following optic nerve stimuli. Jour. Neurophysiol., 1940, v. 3, pp. 308-322.

[10] Marshall, W. H., and Talbot, S. A. Recovery cycle of the lateral geniculate of the nembutalized cat. Proc. Amer. Physiol. Soc., 1940, pp. 121-122.

[11] Talbot, S. A., and Marshall, W. H. Binocular interaction and excitability cycles in cat and monkey. Proc. Amer. Physiol. Soc., 1941, pp. 279-280.

[12] Andersen, E. E., and Weymouth, F. W. Visual perception and the retinal mosaic. Amer. Jour. Physiol., 1923, v. 64, pp. 561-591.

[13] Averill, H. L., and Weymouth, F. W. Visual perception and the retinal mosaic. Jour. Comp. Psychol., 1925, v. 5, pp. 147-176.

[14] Adler, F. H., and Fliegelman, M. Arch. of Ophth., 1934, v. 12, pp. 475-483.

Discussion

Dr. G. L. Walls (Detroit): Need one believe in any multiplication of elements between retina and cortex, considering Clark's recent demonstration of short association-fibers covering a one-centimeter circle around each cortical visual cell? There is no evidence that the number of optic-radiation fibers exceeds the number of optic-tract fibers.

Dr. Talbot: The reciprocal overlap produces a functional multiplication of path at the geniculate, regardless of the structural relations. However Le Gros Clark has just published direct evidence that the radiation fibers do exceed the optic fibers by at least five to one in certain layers of the monkey geniculate, confirming our indirect conclusion from Cajal.

The argument about resolving power is based more on the amount of overlap, which is supported by the recent evidence of O'Leary on the cat's lateral geniculate. The dendrite structure of the monkey's radiation cells, shown by Clark, suggests similar overlap in the primate.

Dr. David Harrington (San Francisco): Does multiplication of the path explain to your satisfaction the phenomenon of visual-field congruity in calcarine lesions as contrasted with asymmetrical fields in the anterior radiation?

Dr. Talbot: I do not think I am competent to answer that question, because I do not know enough about the detailed distribution of fibers in the radiation.

Dr. H. J. Smith: I should like to ask the speaker if he has any evidence on the asserted overlapping of the crossed and uncrossed fibers in the external geniculate body and that they are reversed on the opposite side of the brain. I have forgotten who worked that out, but I found it in the literature. The fibers from the

same geniculate body, the crossed fibers and the uncrossed fibers, were found to locate or terminate in different layers alternately in the external geniculate body; on the opposite side of the brain the exactly reverse order was observed.

Do you find that that is true? Do you find anything to support that belief?

DR. TALBOT: Not directly. We have looked pretty hard for it, but we have not been able definitely to demonstrate with electrodes the specific activities of a single geniculate layer. We are still working on it. Layering seems to be supported by a great deal of anatomical evidence. The picture is confirmed physiologically by the relations of facilitation and inhibition between the eyes, at the geniculate level. Dr. Marshall, would you care to comment on that?

DR. MARSHALL: If I understand your proposition, anatomically there is no doubt about the layer structure.

DR. SMITH: You believe that is true.

Then just one question more: Do I understand you to say that in the retinal localization the upper temporal fibers and the upper nasal fibers are of about the same distribution?

DR. TALBOT: In both cat and monkey the upper temporal fibers from one eye project to the same cortical area as the upper nasal fibers of the other eye.

DR. SMITH: Do you find that the upper and the lower nasal fibers have a corresponding termination in the calcarine area?

DR. TALBOT: The upper and lower nasal fibers from one eye project not to the same but to sagittally adjacent areas on the striate cortex of these animals.

DR. SMITH: Also in the macula, do you find that they terminate toward the internal geniculate dorsally?

DR. TALBOT: The "macular" fibers terminate dorsally on the cat's external geniculate, in our functional observations.

EYE DISEASE DUE TO VITAMIN DEFICIENCY IN TRINIDAD

Tropical nutritional amblyopia; Essential corneal epithelial dystrophy; Conjunctional bleeding in the newborn

Vivian M. Métivier, F.R.C.S. Ed., D.O.M.S.
Trinidad

After reviewing evidence from 48 different colonial territories, the Committee on Nutrition in the Colonial Empire (1939) found that diseases caused by deficiency of vitamin A were perhaps the most common of all. Vitamin-A deficiency is, however, of little importance in Trinidad in the sight-saving work of the ophthalmic surgeon. Keratomalacia is infrequently met with, xerosis of the cornea is rare, and only an occasional case of night-blindness is seen. Bitot's spots of a chronic tropical type prevail among Trinidad-born East Indian children, but the condition does not respond to treatment, and was dealt with by me in another paper, "Bitot's spots in Trinidad" (1941). While frank beri-beri and classical pellagra are clinical curiosities with us, the ocular manifestations of vitamin-B deficiency, B_2-complex syndromes in particular, provide a good deal to think about and do. These syndromes have been the subject of many communications from workers in different colonial territories, and they cause an appreciable amount of physical discomfort, bad health, and partial disability that may be long standing or permanent. Fitzgerald Moore (1940b) has written several papers on nutritional retrobulbar neuritis; he lays great stress on the importance of vitamin-B_2 deficiency as the cause of eye disease in Nigeria, while pointing out the relative scarcity of eye conditions that are the result of avitaminosis A. Without the help of colonial hospital experience at the time, I was able to say, several years ago (Métivier, 1932) that vitamin deficiency was a factor in the causation of blindness in the colony and called attention to obscure disease of the optic nerve. During the past 50 years there has been an interesting sequence of accounts of vitamin-deficiency symptom-complexes that appear to have some common link or chain connecting them. They all have an ocular condition in their symptomatology as shown in table 1.

From my own experience, the eye conditions—photophobia, hazy vision, lacrimation, failure of central visual acuity, and partial optic atrophy—appear to fit into two distinct clinical pictures that are the result of failure of vitamin B_2 to maintain the due nutrition of the cornea and optic nerve, respectively. They may occasionally be found together in the same patient, and the distinctive names, essential corneal epithelial dystrophy and tropical nutritional amblyopia, are suggested for them. The former has not been described before; the latter is the nutritional retrobulbar neuritis of St. John, Moore, and others. From the ophthalmological aspect, each clinical picture has its own individuality and should be differentiated as a disease. Patients who come to the eye departments suffering from either of them can readily be distinguished by the nature of their complaint, their appearance, and the history they give on being questioned. When the optic nerve is affected, the patient complains of severe visual defect; with the corneal lesion, the leading symptom is photophobia.

The syndromes that have been described by Moore, St. John, Browne, Landor and Pallister, and others are not seen in my patients.

TABLE 1

VITAMIN-DEFICIENCY STATISTICS

Author	Year	Syndrome	Ocular Condition	Location	Other Signs and Symptoms
Strachan	1888 1897	Multiple neuritis	Optic neuritis; trophic changes in Cornea; desquammation—at eyelid margins	Jamaica	Burning feet; excoriation at corners of mouth, the prepuce, anus, and vulva
Scott	1918	Central neuritis	Photophobia; loss of visual acuity	Jamaica	Burning feet; stomatitis; diarrhea; numbness and tingling, incoördination and difficulty in walking
Wright	1927 1930 1934	A and B avitaminosis disease of Sierra Leone	Failure or dimness of vision	Sierra Leone	Angular stomatitis; eczema of scrotum; glazing of tongue; disorders of nervous system
	1936	Polyavitaminosis and asulphurosis	do	do	do
Bradley	1929	Décoquée	Visual defect	Seychelles	Soreness at angles of mouth and eyelids; eczema of scrotum or vulva; defects of hearing
Sharples	1929	Burning feet	Impairment of vision	British Guiana	Burning fingers and palms
St. John	1930 1936	Avitaminosis A and B	Retrobulbar neuritis	Barbados	Lesions of mucocutaneous junctions of lips and eyelids, at angles
Browne	1932	Avitaminosis	Retrobulbar neuritis; optic atrophy	British Guiana	Angular stomatitis; sore tongue
do	1936	Vitamin-B deficiency	Retrobulbar neuritis	do	Deafness
	1934	Avitaminosis	Retrobulbar neuritis	Nigeria	Sore tongue and mouth; lesions at angles of eyelids; scaly itchy scrotum
Moore	1937	Nutritional retrobulbar neuritis (vitamin-B_2 deficiency)	Optic atrophy	do	do
	1939	Pellagra	do	do	do
Landor and Pallister	1935	Avitaminosis B_2	Dimness of vision	Malaya	Eczema of scrotum; glossitis; cheilosis; subacute combined degeneration of the spinal cord
Stannus	1936	Pellagra and pellagralike conditions	Photophobia; haziness of vision; loss of central acuity; partial optic atrophy	Warm climates generally	Angular stomatitis; glossitis; burning feet; numbness in fingers and toes; eczema of scrotum
Cohen	1936	Pernicious anemia	Optic atrophy	England	Glossitis; subacute combined degeneration of the spinal cord
Clark	1937	Malnutrition in children	Retrobulbar neuritis	Jamaica	
Oden & Sebrell	1939	Ariboflavinosis		Southern U.S.A.	Angular stomatitis
Kruse	1940	Ariboflavinosis	Keratitis	U.S.A.	Cheilosis; glossitis; seborrheic dermatitis

Sore tongue or glossitis is very rarely met with; a mild degree is occasionally observed on inspection. My patients never complain of their tongues.

Scrotal, vulval, and perineal lesions are never found in our eye cases, but one patient with corneal epithelial dystrophy had the free border of the prepuce affected with an eczematous condition.

Cramps and numbness in fingers and toes are, however, often complained of by vitamin-deficiency patients of our eye departments. The symptoms are troublesome, and difficult to get rid of; and in a few bad cases there has been incoördination and difficulty in walking. Pure vitamin B_1, administered parenterally, has recently given much better results.

With the help of observations and suggestions made by Manson-Bahr (1940) and Spies (1939-40) in recent work, table 2 has been prepared:

me, in both corneal and optic-nerve nutritional-disease patients, is a form of *dry parched skin* that is not phrynoderma. Patients who show it to well-marked degree are often obviously malnourished.

Angular stomatitis, first described by Stannus (1912), has had many different names applied to it in different parts of the world, and in Trinidad it is popularly known as "wappia." School children hide the white sodden patches with ink dabs, and I have seen boot polish used by an adult. The condition is of special interest to ophthalmic surgeons because the internal and external angles of the eyes are often affected, and there are occasions when the mouth lesions may be barely noticeable. The condition has been very

TABLE 2

THE KNOWN VITAMIN-DEFICIENCY FACTORS INVOLVED IN THE PRODUCTION OF DIFFERENT CONDITIONS

Vitamin Factor	Condition Caused by Deficiency	Syndrome
B_1		Beri-beri
B_1	Neuritic signs	Pellagra
B_1?	Numbness in fingers and toes; burning feet	Pellagra; tropical nutritional amblyopia
B_2 complex	Glossitis and rarely changes in spinal cord	Tropical sprue
B_2 complex	Glossitis and changes in spinal cord	Addisonian pernicious anemia
B_2 complex	Glossitis, stomatitis, megalocolon	Idiopathic steatorrhea
B_2 complex	Defective nutrition of optic nerve (failure of central acuity; partial optic atrophy)	Tropical nutritional amblyopia (retrobulbar neuritis)
B_2 complex	Photophobia, hazy vision	Essential corneal epithelial dystrophy
Riboflavin (or Lactoflavin)	Angular stomatitis (cheilosis), lesions at canthi and naso-labial folds, cheilitis	Pellagra; tropical nutritional amblyopia; essential corneal epithelial dystrophy
Nicotinic acid ("P.P. factor")	Dermatitis, gastro-intestinal and mental symptoms	Pellagra
Nicotinic acid	Eczema of vulva and scrotum	Pellagra; tropical nutritional amblyopia

Lesions of the central nervous system have not been found by hospital physicians when these cases have been referred to them.

"Burning feet" is often elicited on questioning, and patients with it say that they are unable to walk about with bare feet. As in British Guiana, it is a common complaint of East Indians on sugar-cane estates. A sign frequently observed by

fully discussed in medical journals during the past year, and excellent photographs of it have been published by Wright (1930) and Moore (1937, 1939). They show the heaped-up epithelium, white and sodden, at the canthi, and fissure formation in the adjoining skin, which is often darkly pigmented at the external commissures with a collection of seborrheic and epithelial débris. It clears up quickly

with vitamin-B_2 preparations, and Oden and Sebrell (1939) first called the condition ariboflavinosis. I have seen cases recover in 10 days with marmite and an improved diet.

Photophobia in vitamin-B deficiency is a true fear of light and is very rarely reflex blepharospasm. Patients with it have difficulty in using their eyes in the hot sun and see better when the sun begins to set.

Tropical nutritional amblyopia. The name given in the heading is chosen for the reasons that follow. Changes that have their origin in vitamin-B_2 deficiency are not primarily inflammatory in type, and optic-nerve affections would be expected to correspond to those that occur in other tissues. Defective nutrition of the optic nerve with failure of central acuity of vision is a tropical disease; and the symptomatology resulting from it seems to correspond more closely to that of the toxic amblyopias. It may be that the ganglion cells of the retina rather than the optic-nerve fibers are primarily involved. In all this there is a wide field of interest waiting to be fully explored by those who may be granted facilities and equipment with which to attempt it. It is worth noticing that there have been no communications on vitamin deficiency as the cause of *acute* retrobulbar neuritis; while, apart from toxic amblyopia, chronic retrobulbar neuritis is a rare disease. Acute retrobulbar neuritis, acute axial neuritis, is very seldom seen by me in colonial hospital and private practice in Trinidad, and I have had only five cases over a 10½-year period. At the same time, disseminate sclerosis is never known to develop in the colony. Syphilis, however, plays a very important part in the production of optic-nerve lesions—that is, papillitis, neuroretinitis, consecutive optic atrophy, retinitis proliferans, and, last and least, simple or primary optic atrophy (Métivier, 1937)—while syphilitic eye disease is our commonest cause of blindness (Métivier, 1932). In 1931, in Barbados, St. John, Browne of British Guiana, and myself agreed that syphilitic infection was a disturbing factor in the British West Indies when attempts were made to define the role of vitamin deficiency in the causation of optic-nerve affections. The fact that retrobulbar neuritis, directly due to syphilis, is a rare disease (Traquair, 1933, 1935) does not altogether help to define the clinical picture. Moore and Woods (1940), in an exhaustive study of recent work on syphilitic optic atrophy, came to the conclusion that the most promising approach to the problem is to regard syphilitic optic atrophy as the result of infection and vitamin deficiency. Only a limited number of persons with late syphilitic infection develop optic atrophy or any form of nerve lesion, and the problem presented by that fact is even more pronounced with us in Trinidad because syphilitic infection is heavy while tabes and G.P.I. are exceedingly rare diseases. Further, the great majority of our patients who suffer from optic atrophy, in both the partial and complete form, have positive blood Wassermann reactions and Kahn tests.

It is my experience that, in Trinidad, primary, simple, or tabetic optic atrophy is a rare manifestation of syphilis, and when the whole optic-nerve head shows atrophic changes they can usually be found to be of a consecutive or secondary type, the result of "interstitial" syphilis. These patients, unless treated in the very early stages, go on to blindness. With partial atrophy, involving from a little less than the temporal half of the optic disc up to nearly three fourths of the whole papilla surface, the upper nasal quadrant always being spared, vitamin-B_2 deficiency is the important etiological factor. With this lesion, patients rarely

become certifiable as blind.

There are three features to consider in explanation of the discrepancy of reports on B_2-deficiency syndromes. In the first place, several observers had patients who were inmates of boarding schools, prisons, camps, and other institutions where the diet would be, more or less, the same for each inmate. In Trinidad and other West Indian Islands the patients are of the hospital class, free to choose their own diet in their homes. Second, a diet composed of milled cereals and starchy roots is deficient in the B_2 group of vitamins; and components of the B_2 complex that are lacking in the diet may vary according to the nature of the staple and supplementary foods in the community. Also, diets deficient in vitamin B_2 also tend to be deficient in other vitamins; and a diet lacking in one factor of the B_2 complex is likely to be insufficient in others (Aykroyd and others, 1939). Finally, discrepancies in the symptomatology described by different workers are likely to arise from differences in race, climate, tribal custom, community habits, and medical and social conditions between one colony and another, not necessarily widely separated.

Illustrative case records with commentaries

Case 1. R., an East Indian male, aged 28 years, complained on December 20, 1940, of failure of vision, right eye, of six days' duration, with slight pain. His vision was, R.E. perception of hand movements; L.E. 6/6. He was healthy and strong, well nourished, with no signs nor symptoms of avitaminosis. There was slight edema of the retina around the disc of the right eye, with blurred disc margins. The fundus of the left eye was normal. His blood Wassermann reaction was negative, but the Kahn reaction was positive. Bemax and marmite were ordered for him on his admission to hospital the same day, and he was given protein-shock therapy with milk injections. On December 24, 1940, the vision of the right eye had improved to 6/24 and a month later it was 6/9, with very slight pallor of the disc remaining. On January 21, 1941, he resumed employment. My diagnosis was acute retrobulbar neuritis of indefinite etiology.

Case 2. L. L., a policeman, aged 38 years, was sent to me on the 1st of January, 1941, for refraction because he said that he had difficulty in reading, especially at night. He said that his sight had been getting gradually dimmer for several months. His vision was, R.E. 6/60; L.E. 6/36. There had been slight pain in the right eye three months previously. He did not smoke nor chew tobacco. He had a well-marked central scotoma for red and green, and his fundi were normal. I admitted him to hospital and put him on bemax, marmite, and livogen, as well as protein-shock therapy, using T.A.B. vaccine. With the latter he had very good pyrexial reactions. He had no signs nor symptoms of avitaminosis, although his diet was unsatisfactory, and he was fed from his home, a few miles out of town, by carrier. In hospital he was given eggs, raw tomatoes and carrots, lettuce and citrus fruit, in addition to the ordinary diet. He made a very good recovery and on the 15th of January, 1941, his vision was 6/6 nearly, with each eye. On the 4th of January, 1941, his blood Wassermann reaction was reported positive and the Kahn test negative. There was a history of antisyphilitic treatment by injections some years previously. I believe this case to be one of early tropical nutritional amblyopia.

Case 3. A. H., a male East Indian, aged 24 years, was sent to me on the 28th of January, 1941, by the medical officer of a sugar company for a report on the con-

dition of his eyes. The patient had applied for the post of tool clerk at the sugar factory. He was found to have a form of conjunctival injection that I call "rosy eyes." It is a type of apple-pink injection of the ocular conjunctiva exposed in the interpalpebral area, with dilated vessels that run into the limbus. This man's vision was 6/6 with each eye and 6/5 with both eyes together. He was a strong, well-built man who always worked indoors, and he played cricket and football throughout the year. There was no secretion of mucus and no conjunctivitis. He did not complain of photophobia and there was no lacrimation. I have seen the same condition in two female patients who were suffering from essential corneal dystrophy, and it cleared up with vitamin-B therapy in both cases. It is most likely due to vitamin-B_2 deficiency and would appear to be related to the circumcorneal injection of ariboflavinosis that has been described by Kruse and others (1940), Sydenstricker and co-workers (1940).

Case 4. G., an East Indian woman of 25 years, complained on January 9, 1940, of irritation and watering of the eyes in strong sunlight of about a week's duration. She said that her vision was not affected. No abnormality was found on examination of her eyes. Her baby, one year old, was still being nursed. There was slight angular stomatitis with marked dryness of the skin and hair. She was given cod-liver oil and bemax, the baby was weaned, and all her symptoms were relieved in a fortnight. Many negro and colored school children who have normal eyes come to the eye clinic with similar symptoms, and say that they are unable to read without discomfort. There is usually an absence of refractive error, and treatment with vitamins A and B brings about relief.

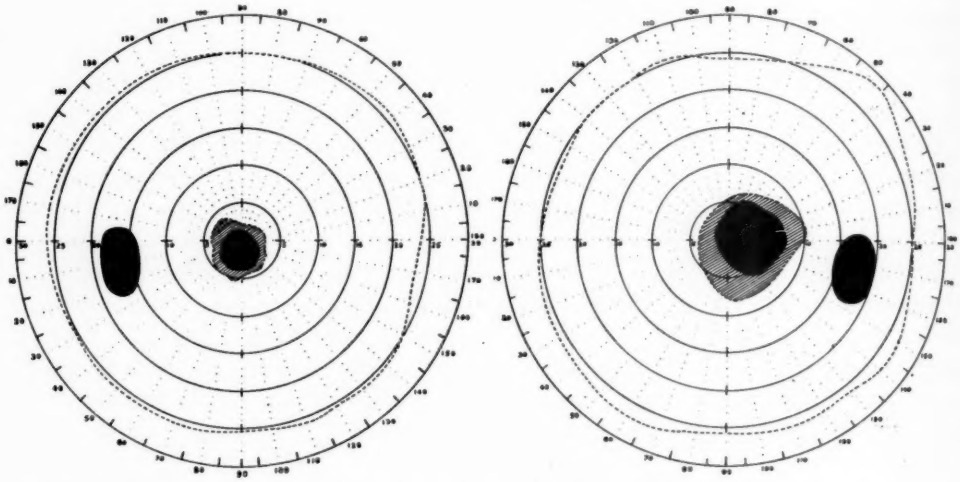


Fig. 1 (Métivier). Case 5. Left, visual field of left eye, showing central scotoma for 1/2,000 white; slightly larger for 10/2,000 red. Peripheral isopter, 2/2,000 white. Blind spot 30/2,000 white. Peripheral field normal for 1/330. Vision 6/18 nearly, January 22, 1940.

Right, Visual field of right eye. Central scotoma for 1/2,000 white; larger for 10/2,000 red. Eccentric position of blind spot. Tropical nutritional amblyopia. Vision 6/36 on January 15, 1940.

Case 5. E. C., a negress, aged 19 years, complained on January 2, 1940, of six weeks' failure of vision and inability to read. Her general condition was good, but angular stomatitis was present and had developed after the birth of a baby now

eight months old and breast fed. At 8½ months, the baby weighed 21 pounds. There was a history of one miscarriage. Vision was R.E. 6/60; L.E. 6/36; The fundi were normal; there was a well-marked central scotoma for red and green, as is shown in figure 1. Peripheral fields were normal. There were early signs of corneal epithelial dystrophy in the left eye. The patient was admitted to the hospital and put on cod-liver oil, bemax, and marmite. Wassermann and Kahn tests were both negative. After 10 days' treatment the angular stomatitis was nearly all gone. From January 3 to 12, 1940, she was given protein-shock therapy, with good pyrexial reactions. On January 29th her vision had improved to R.E. 6/36; L.E. 6/18. She was then given Betaplexin (Winthrop Chemical Company, Inc.) in place of other vitamin-B preparations. On February 2d, when made an outpatient, the corneal epithelial dystrophy was cured; and on the 30th of March she said that her vision was again normal. She could then read 6/6.

Case 6. L. A., a negress, married, aged 26 years, was sent to me by the D.M.O. of a Tobago District on the 30th of November, 1939. A fine, big strong woman, her vision had failed since the birth of a baby six months previously. Mother of six children, all alive and well, she had had no miscarriages. She had been given six bismuth injections without improvement. There was a history of numbness and cramps in the lower limbs as well as burning feet. Vision was R.E. 5/60; L.E. 3/60. The peripheral visual fields were intact, and she got about in a normal manner. Both optic discs had slightly blurred margins. There was corneal epithelial dystrophy, both eyes, and more marked in the left. Wassermann and Kahn tests were both negative, with two separate blood examinations. The patient was admitted to the hospital and put on bemax, marmite, and yeast together with a high-vitamin-containing diet. From the 30th of November, 1939, to the 14th of December, 1939, she was given protein-shock therapy, with good pyrexial reactions. After one month's treatment, her condition remained unchanged; bemax was stopped and Benerva (B_1) tablets substituted. They were given for 11 days, the limit of my stock. She had carious teeth extracted. She left hospital on the 31st of January, 1940, without any improvement in her vision, but the cramps and burning feet were relieved and the corneal dystrophy had cleared up. She failed to keep under observation by the D.M.O. on her return home. (More than one vitamin-B preparation is often ordered because some patients dislike marmite, and at times our stock of any one preparation may run out.)

Case 7. Mc. S., a negress, aged 26 years, complained on the 4th of January, 1940, of two months' failure of vision. Her first baby had died at the age of 15 days; there had been one miscarriage three years ago; one child was two years old, and a breast-fed baby nine months old. At 10 months the baby weighed 19 pounds. The patient's vision was 6/60, each eye, and there was essential corneal dystrophy in both, the affected epithelium staining well with Bengal red. There was a central scotoma for red and green, with good peripheral fields and normal fundi. The blood Wassermann and Kahn tests were both positive. The patient was put on bismuth injections and cod-liver oil and bemax. She was given dietetic advice and told to take Fleischmann's yeast at home. The baby was weaned. On the 15th of February, 1940, her vision was a little better than 6/60 with each eye, and nearly 6/24 with both eyes. Her vision finally improved to 6/12 each eye, 6/9 with both together, and the corneal epithelial change disappeared.

Case 8. Q. S., a negress, married, aged 32 years, reported on the 20th of December, 1939, with a history of four months' failure of vision. She had four healthy children, the youngest, seven months old, breast-fed, weighing 18½ pounds. She was a fine healthy-looking, buxom woman with no symptoms of vitamin deficiency. Her vision was, R.E. 6/60; L.E. 6/60 with well-marked central scotoma for red and green, 5 degrees around and including the fixation point with 8.6-minute visual angle and red test object. Peripheral fields were normal; ophthalmoscopic and all other examinations negative, including Wassermann and Kahn tests. There was no response to treatment in hospital during a month with a rich vitamin diet, bemax, marmite, and protein-shock therapy. She then became an out-patient for five weeks, but no improvement in vision resulted. This woman's central acuity will remain unchanged together with pallor of the temporal half of each optic disc. Her partial incapacity will not be readily noticed because she can get about in a normal manner. Two negro school teachers suffering from the same affection struggled along for several years with vision 6/60 and 6/36, respectively. Convex lenses with prisms, bases in, had helped them; but one could not accept promotion and the other was ultimately forced to resign.

Case 9. C. H., a negro boy of 13 years, was seen on the 13th of July, 1939, because of inability to read at school. Although his diet was inadequate, his general health was good, and he had no signs of vitamin deficiency. His vision was R.E. 6/60; L.E. 6/24; his fundi were normal. The blood Wassermann and Kahn tests were both negative. After treatment as an out-patient, he was admitted to hospital on the 24th of August, 1939, and given marmite, bemax, and cod-liver oil, as well as a high-vitamin-containing diet. He also had protein-shock therapy. No improvement in vision resulted. Discharged from hospital on the 13th of September, 1939, he continued as an out-patient. On the 1st of February, 1940, his vision was R.E. 6/60; L.E. 6/60; the temporal half of the disc of the right eye was then very pale, while that of the left was pale. Convex lenses with prisms did not help him to read, and he was forced to leave school. He may possibly be an unusual case of Leber's disease.

Case 10. A. J., an East Indian woman, married, aged 23 years, complained of three weeks' failure of vision on the 20th of December, 1939. Although her diet was very unsatisfactory, her general condition was good, and she was nursing a baby eight months old, weighing 21½ pounds. The baby was a fine child, healthy and sturdy. One other child had died when 17 days old. Her vision was R.E. 6/36; L.E. 6/36. She had numbness in her fingers and burning feet. Her fundi were normal. Blood Wassermann and Kahn tests were negative. With dietetic advice, weaning of baby, and cod-liver oil and bemax her vision improved to 6/12 with each eye within a month. She then continued in the care of the D.M.O.

Case 11. S., a male East Indian, aged 25 years, was admitted to the Eye Ward on the 4th day of December, 1939, from the casualty department, with a history of injury to the left eye, 10 days previously, from a piece of wood. There was a shallow ulcer in the center of the cornea of the left eye and a xerotic looking appearance in the center of that of the right. The patient had angular stomatitis, a very dry parchmentlike skin, and he was unsteady on his legs. He held on to the bed rail when standing up. He complained of numbness in his fingers and pains in his lower limbs that prevented sleep at night. In consultation, hospital physicians diagnosed the limb condition peripheral neu-

ritis. His attitude was simple and his manner childish, but he beat all the other male patients at card games. The angular stomatitis was cured in 12 days, and the corneal xerotic lesions healed rapidly, leaving a faint nebula in the cornea of the right eye, and in the left a shallow facetlike scar. On the 20th of December, 1939, essential corneal epithelial dystrophy was seen to be present in both eyes after examination with high magnification. The epithelial changes radiated from the healed scars. Up to that time the patient had had cod-liver oil, bemax, marmite, and high-vitamin-containing foods. On the 21st of December, 1939, he was given Benerva (B_1 Roche) tablets in place of marmite and bemax. The limb pains continued to be troublesome, and on the 27th of December, 1939, he was given Benerva by intramuscular injection, 1 c.c. daily. After 12 injections the limb pains were greatly lessened. The dry skin had not entirely cleared up and could be made out up to the time he left hospital on the 3d of February, 1940. The corneal epithelial dystrophy was then altogether gone, and his fundi were normal. He could stand up without help and walked about fairly well. The patient failed to continue under observation as an out-patient. The dry parched condition of skin and hair that this man suffered from is frequently seen by me in vitamin-B deficiency patients in the eye departments. It differs from phrynoderma; and I believe that it is due to B_2 deficiency, although it does not respond promptly to treatment with marmite. Further investigation with pure B_2 factors is needed in these cases.

Case 12. M. M., a male aged 20 years, was referred to me on the 28th of January, 1941, by the M.O. to a Sugar Estate Company for a report on his eyes before employment. His vision was 6/36 with each eye, and he said that his vision had begun to fail one year ago. He had angular stomatitis, but his scrotum, perineum, and tongue were normal. The only eye changes found were pallor of a little more than the temporal half of the disc of the right eye and pallor of about five sixths of that of the left, the upper nasal quadrant showing the least change. He was admitted to hospital and given 4 mg. of lactoflavine daily, 1 mg. by injection.

That was for four days; and for five days after that he had 5 mg. daily with 2 mg. intramuscularly. This man was 5 feet 8 inches in height and weighed 144 pounds. He was also given bemax, marmite, and livogen, with eggs, raw tomatoes, citrus fruit, and green vegetables as special additions to his diet. On the 14th of February, 1941, his vision was R.E. 6/24, L.E. 6/60. Between the 11th and the 14th of February, 1941, he had 3 mg. lactoflavine daily by mouth. On the 14th of February he was discharged from the hospital; he continued treatment with bemax and marmite at home. On the 28th of February his vision had improved to 6/18 each eye and nearly 6/12 with both eyes together. He was then allowed to go to work, but he failed to continue treatment. I sent for him on the 25th of March, 1941, and found that his vision had then improved to 6/12, each eye, and 6/9 with both eyes together. The pallor of each disc was then much less marked and was limited to the temporal half of each optic-nerve head. The response to treatment in this case is quite exceptional and suggests that the lactoflavine (riboflavin) was primarily responsible for it.

Essential corneal epithelial dystrophy

Definition. This condition was first described under the title, "A 'new' eye disease," in the 1934 Annual Report of the Surgeon General, Trinidad and Tobago. In a published article (Métivier,

1937b) I made further reference to it under the subhead, "Cases of corneal dystrophy in which the blood Wassermann reaction was positive." The first case was seen soon after my appointment to the Colonial Hospital, San Fernando, in May, 1932; and four patients were found to have the condition that year. Altogether, 161 cases have been under my care there up to the end of 1939. Two patients were seen in private practice; and at the Colonial Hospital, Port-of-Spain, 29 patients have been treated, making a total of 192 cases in Trinidad to the end of 1939. Counting only hospital patients, the incidence is 190 in 13,667 new eye cases or 1 in 71.9.

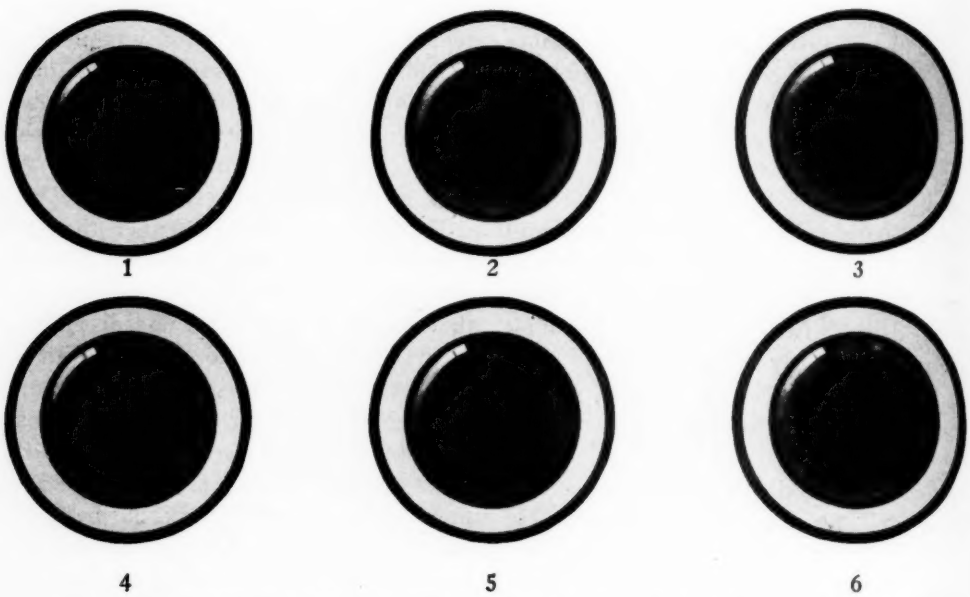


Fig. 2 (Métivier). Semidiagrammatic drawings to show lesions in essential corneal epithelial dystrophy (×9). Executed by Theodore Hamblin, Ltd., London.

The name, essential corneal epithelial dystrophy, was chosen to distinguish the condition from other dystrophies of the cornea previously described, especially that popularly known as Fuchs's epithelial dystrophy. It is a condition in which a geometrical arrangement in a double line, running transversely across the cornea, at the level of the lower part of the pupillary area, shows a faint grayish-white disturbance of the epithelium which is made up of fine points like dots and commas. At times the double line is incomplete, but minute prolongations usually extend above and below it to make up a coral-shaped pattern lying as if in one place. The affected area stains well with fluorescein or Bengal red, taking up the stain quickly, as a rule. In typical cases only the linear arrangement is seen, with prolongations that run to points in a triangular fashion; but in patients with obvious signs and symptoms of malnutrition the whole corneal surface, with the exception of the periphery, is found covered with faint grayish-white points of epithelial haze, and in that way the double transverse line of the interpalpebral area is obscured. The presence of a nebula also affects the distribution of the pathologic pattern, and drawings are shown in figure

2 to illustrate the different appearances. The lesion is never accompanied by signs of inflammation, conjunctival or ciliary injection even in the form of a faint blush. It is a dystrophy due to avitaminosis B_2, and most likely deficiency of riboflavin (lactoflavin). When relapses have taken place, or the condition has been long standing light brownish feathery lines replace the original pattern; and that condition has been seen to disappear under prolonged treatment.

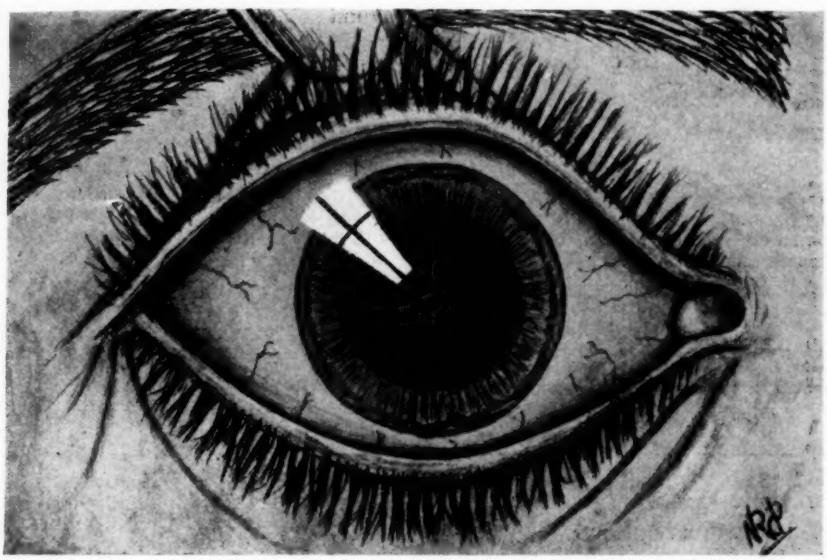


Fig. 3 (Métivier). The eye in No. 5 (× 9) of figure 2.

Etiology. The affection in its purest form is a sequela of avitaminosis B_2—ariboflavinosis predominantly—in nursing mothers who usually appear quite fit and strong, well covered with flesh, and often with no symptoms except those related to their eyes. Both East Indians and negroes are affected and occasionally a Chinese woman. A few husbands and many children of affected mothers have been examined with negative results. The babies are always fine, healthy children, often above the average weight for their age. Two mothers had breast-fed twins, and another was nursing a baby three months old while she had thriving triplets, a year and nine months of age. Suckling is nearly always prolonged far beyond the recognized period, without any regular or substantial addition to the diet of infants as old as a year. With mothers who have stopped breast feeding the history very often dates back to late pregnancy, the birth of a baby, or a period of suckling. Faulty dieting is the primary cause, and poverty is an important contributory factor. Environment has sometimes been the cause of an indifferent choice of articles of diet; but lack of appreciation of food values, particularly as regards vitamin content, is the main reason for a deficiency of vitamin B_2 in the diet of Trinidadians. Many working-class mothers have severe demands made on their individual economy. One East Indian woman, aged 36 years, took 10 months to get better. She had six children, the eldest, 14 years of age, and she was nursing a baby three months old. She looked after an ailing husband, did the house-

hold work, milked a cow, and sold the milk as an itinerant vendor, carrying the milk pail on her head. She always fell sound asleep in the out-patient waiting room. In spite of all advice, she continued to suckle her child until it was a year old. Other signs and other symptoms of vitamin deficiency are invariably present, and in order of frequency are as follows: angular stomatitis, dry parched skin, burning feet, numbness in hands and feet, pallor or atrophy of temporal

Symptoms. The condition will be strongly suspected from the symptoms complained of. Photophobia is the leading one: "My eyes water and burn, and when I look at anything afar off in the hot sun I cannot fix it. My sight has lately also become a little misty, but when it cools off in the evening I can see much better." When the patient's first complaint is defective vision, a central scotoma or partial optic atrophy, due to nutritional amblyopia, is always present as a

TABLE 3

AGE PERIODS OF 192 PATIENTS

Group	Age Period	Age Limits	Total Number
Nursing mothers	16 to 45	16 to 40	57
Women not recorded as Nursing mothers	16 to 45	16 to 43	52
Other women	46 and over	46 to 60	5
Girls	under 16	10 to 14	5
			119
Males	16 to 45	17 to 45	56
Males	46 and over	46 to 70	17
Boys	under 16		0
			192

half of optic disc, phrynoderma, sore tongue, incoördination and difficulty in walking, "rosy eyes" rarely, and xerophthalmia very rarely. More patients are seen during the hot dry season of the year than during the period of rains and cloudy days.

Pathology. Vitamin B_2, which is closely related chemically to Warburg's yellow enzyme, serves as an oxidizing agent to the tissues, doing so by breaking down oxyhemoglobin to hemoglobin. Since the cornea is avascular no hemin substances are normally present in it, and oxidation within the cell is accomplished by the enzyme directly. Disturbance of the functioning of this enzyme system results in dystrophy of the corneal epithelium at the site where the eyes are most exposed to direct sunlight, because vitamin B_2 is biologically inactivated by light.

complication. Reflex blepharospasm is never present, but some patients appear to weep when relating their distress, and rarely profuse watering of the eyes takes place during examination. In well-established cases vision is usually reduced to about 6/12 Snellen with each eye.

Diagnosis. If suspected, the condition may be diagnosed with Hamblin's desk inspection lamp and a Bishop Harman loupe. With a plus 12 lens behind the sight hole of the self-luminous ophthalmoscope the double line in the cornea can be made out; and that was the manner in which the lesion first came to my notice. To see the appearances as represented semidiagrammatically in figure 2, examination by oblique illumination and a corneal loupe is necessary.

Various forms of keratitis and epithelial dystrophy of the cornea have been

described in recent years, but none of them appear to be the same condition. They are "A case of dystrophy of the cornea which disappeared on the administration of fats" (Langdon, 1928); "The mild form of epithelial dystrophy of the cornea" (Gifford, 1932); "Corneal epithelial changes" and "Epithelial degenerations" (Basil Graves, 1936); "Some cases of epithelial dystrophy, Keratitis" (Morgan, 1937); "Keratitis ramificata superficialis" (Wille, 1938); and "Ocular manifestations of ariboflavinosis" (Kruse *et al.*, 1940). The signs and symptoms in Langdon's case appear to correspond in many respects to those of my own cases, but he inclines to the view that the condition he describes in Fuchs's epithelial dystrophy.

Prognosis is good, patients recovering as a rule after 5 to 12 weeks of treatment; but two patients with well-marked signs recovered in two weeks with doses of 5 mg. of lactoflavine (riboflavin) daily, 2 mg. by intramuscular injection and 3 by mouth. In East Indians the return to normal of the corneal epithelium takes longer than in negroes because of their habits of diet. Relapses have taken place in a few patients, usually East Indian women, after periods of a year or so.

Treatment consists primarily in the administration of vitamin B_2 and protection of the cornea with smoked glasses. Bemax and marmite have been used, they being the only medium of supply generally available at the colonial hospitals for out-patients. Vitamin A has been given also in a 50-percent cod-liver-oil emulsion, but no patient has recovered on cod-liver oil alone. A few out-patients have been able to buy yeast for themselves. In-patients have been given bemax, 0.5 ounce daily; Fleischmann's yeast, 1 cake daily; and marmite 0.5 ounce daily together with an improved diet, yet they have always failed to recover under five weeks. Nicotinic acid was given in full doses to two patients without any change taking place in the corneal condition. Vitamin B_1 did not effect a cure in three patients, but they all asked for more of it when they found that their limb pains were lessened. Lactoflavine (riboflavin) was first tried with a very small supply kindly sent to me by the Jamaica agents of British drug houses through their local representatives; but in August, 1940, a further supply, kindly given to me by the same firm, was lost through enemy action. Early in 1941 all the lactoflavine available in the colony was obtained but it was only enough for two patients, E. L. and P.; and response to treatment in both cases was admirable. Patients have, on the whole, taken remarkably well to the slower rate of recovery with bemax and marmite. The nature of their complaint is explained to them, its relationship to diet pointed out, and a good prognosis is given with assurance. And so, they report regularly as out-patients and return for treatment at once if symptoms recur long afterwards.

Cases treated with lactoflavine (riboflavin)

E. L., a fine healthy-looking negro girl of 14 years, came to the eye clinic on the 15th of January, 1941, complaining of photophobia and watering of her eyes for 2½ months. She was then unable to do her school work. Ink dabs hid her angular stomatitis and there were small patches of follicular hyperkeratosis on her elbows and knees. Her vision was 6/9, each eye, and there was the well-marked double line of corneal epithelial dystrophy in both eyes. Her weight was 121 pounds but her diet was very unsatisfactory, being very poor in vitamin B and inadequate in vitamin A. She was told to continue with her usual diet; and 5 mg. lactoflavine was given to her daily, 2 mg. by injection and 3 by mouth. With five days of that

treatment the angular stomatitis disappeared; and after two weeks, in all, the corneae became normal, with vision returning to 6/6. On the 12th of March, 1941, the patient was put on prepalin, a very high vitamin-A-content preparation made by Glaxo, Ltd., for treatment of the skin lesion. When the corneal lesion had cleared up dietetic advice was given.*

P., an East Indian woman of 29 years, complained on the 21st of January, 1941, of watering of her eyes in the hot sun and hazy vision. She was nursing a fine, healthy baby 22 months of age and she did field work, heading Para grass on a sugar-cane estate. She had three children, and her husband was sickly. She had no sore tongue, burning feet, numbness, nor cramps. The lower halves of both corneae showed very well-marked diffuse changes of corneal epithelial dystrophy, except at the periphery, the typical double line of the pupillary area being obscured. On each side of the limbus, the temporal aspect only of the interpalpebral area, there was a mild degree of "rosy eyes." Dietetic advice was given, she was told to wean the baby, and bemax and marmite were ordered for her as an out-patient. On the 31st of January, 1941, the corneal condition showed no change, and the patient was admitted to hospital and given riboflavin, 2 mg. daily by injection and 3 mg. daily by mouth. On the 11th of February, 1941, both corneae had improved considerably and on the 18th of February no epithelial change could be observed. The rosy-eye condition was nearly all gone at the same time. Two other patients who began treatment, with bemax and marmite only, at the same period as P. had not made complete recoveries on the 21st of March, 1941.**

* This patient was treated for follicular hyperkeratosis, up to June 28, 1941, with Prepalin and then Adexolin, high-content vitamin-A preparations given me by Glaxo Ltd. She recovered completely. The chronic type of Bitot-spot formation does not respond to treatment with the same vitamin-A preparations.

Conjunctival bleeding in hemorrhagic disease of the newborn

In a letter to The Lancet (Métivier, 1937a) an account was given of the first case seen; and that followed the publication of a complete study of 61 cases (Capon, 1937) in which the conjunctiva was not mentioned as a site of bleeding. In 1938 four babies came to my care and one so far in 1941, but none in 1939 and

** Further examples of results of treatment with riboflavin:

R. M., unmarried East Indian woman of 20 years, reported with angular stomatitis and essential corneal epithelial dystrophy on June 18, 1941. She was treated with riboflavin from June 19th to July 9th, and was given 5 mg. daily, 2 mg. by subcutaneous injection and 3 mg. by mouth. The angular stomatitis cleared up in 5 days and the corneal lesion was healed in 21 days. She had her usual diet at home while under treatment.

S. E., negro workman of 26 years, complained on July 8, 1941, of a cloud over both eyes of 4 to 5 months' duration. He was a well-built muscular man; his vision was R.E. 1/60, L.E. 3/60. The cornea of the right eye had an old scar covering part of the pupillary area, the result of a hypopyon ulcer six years previously—I had performed a Saemisch keratotomy for him with good results. He now had well-marked angular stomatitis, and essential corneal epithelial dystrophy was typically present in the left eye. The nebula on the right side prevented its typical development. The poor vision together with temporal-disc pallor and central scotoma pointed to tropical nutritional amblyopia. He was admitted to hospital and given 10 mg. riboflavin daily, 1 mg. by injection and 9 mg. by mouth, from July 11th to 18th. On July 12th the blood Wassermann reaction and Kahn test were both reported positive and he was put on weekly bismuth injections. During the whole period in hospital, from July 8 to 25, 1941, he had a well-balanced diet as well as bemax and marmite. The latter as well as the bismuth injections were continued when he was an out-patient. On July 18th the angular stomatitis was cured and the corneal lesion nearly all gone. His vision had then improved to R.E. 6/60, L.E. 6/36. He returned to heavy work on August 1, 1941 and on August 19th his vision was R.E. 6/36, L.E. a little better than 6/12.

1940. Three babies were born at home and afterwards admitted to eye departments; the other three were born in hospital. Four babies had bleeding from the conjunctiva only and two from the umbilicus as well. In two the bleeding from the eyes was slight and ceased spontaneously; in the other four it was copious and continuous, and three of them made good recoveries with intramuscular injections of whole blood. The other baby died exsanguine because of delay in treatment. One baby suffered severe damage to the corneal epithelium of both eyes from attempts to stop the bleeding with local applications of adrenalin, swabbing, and bandaging. It is imperative not to make attempts at local treatment for these babies, and one or two have been sent to us as suffering from ophthalmia neonatorum. When the bleeding begins to stop after an intramuscular injection of human blood, liquid paraffin drops are helpful to soften and set free fibrinous masses that adhere to the conjunctiva. Work and progress with vitamin K, fully summarized in the literature (The Lancet, 1939; B.M.J., 1939), pointed out that deficiency of this vitamin is regarded as the likely explanation of the hemorrhagic disease of the newly born, for deficiency of prothrombin in the newborn baby is then most marked. But more recent work on vitamin K (Lancet, 1941) has not yet cleared up the whole story of the pathogenesis of the condition.

Discussion and Summary

The differentiation and synthetic preparation of vitamin-B components, vitamin B_1 and the B_2-complex—vitamin G, riboflavin or lactoflavine; nicotinic acid or its amide; vitamin B_6 or pyridoxine; pantothenic acid—have been accompanied by regular relays of progress in clinical research, and many accounts of disease due to deficiency of vitamin factors have been published by workers in the colonies. "Much of the research which has been undertaken in the tropics has been of an individual nature" (Moore, 1940a), the pathological laboratory being generally regarded as the official agency for colonial research. At the same time, a reckoning of **"vital statistics"** and **"communicable"** diseases continues to provide the argument for repeating that "the general health of the colony remained satisfactory." But, what of the large number of morbidity states now truly presented in terms of vitamin-deficiency disease? For one thing, we see fervent attempts being made to include them all in the symptomatology of pellagra; and "vital statistics" may continue to hold social and economic sway so long as the terms pellagra *sine* pellagra, pellagra *fruste*, and prepellagra are regarded as having any meaning in the light of recent advances.

In this paper closely related syndromes in which eye changes and visual defect are prominent features in warm climates have been considered and discussed in relation to ocular manifestations of vitamin deficiency in Trinidad. The title, "Tropical nutritional amblyopia," is suggested for the disease which results from failure of vitamin B_2 to maintain the due nutrition of the optic nerve, either directly or through retinal elements. When the nutrition of the cornea suffers from deficiency of vitamin B_2, ariboflavinosis in particular, essential corneal epithelial dystrophy is the disease which develops in Trinidad. It is claimed that the description given to this morbidity state is original. "Rosy eyes" are mentioned in relationship to the corneal vascularization recently described as a sign of ariboflavinosis by North American observers. A close watch is being kept for evidence of this affection in Trinidad. Special attention is called to a dry parched-skin condition which is seen in patients suffering

from eye disease due to vitamin-B_2 deficiency.

No reference has been found in the literature to conjunctival bleeding in neonatal hemorrhage. The ophthalmological importance of this vitamin-deficiency state is pointed out, and an account is given of babies treated in our eye departments.

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References

Aykroyd, W. R., Krishman, B. G., and Passmore, B. M. Brit. Med. Jour., 1939, v. 2, p. 825.
Bradley, J. T. Ann. Rep. Med. Dept. Seychelles, 1929 (quoted by Stannus, 1936).
British Medical Journal, 1939, v. 2, p. 1232.
Browne, J. A. Brit. Guiana Med. Annual, 25th year, 1932, p. 25.
———. Caribbean Med. Jour., 1939, v. 1, p. 218.
Clarke, L. M. 1935-36. Quoted by Fitzgerald Moore, 1937.
Cohen, Henry. Lancet, 1936, v. 2, p. 1202.
Gifford, S. R. Arch. of Ophth., 1932, v. 7, p. 18.
Graves, B. In Berens's, The eye and its diseases. Phil. and London, W. B. Saunders Co., 1936.
Kruse, H. D., Sydenstricker, V. P., Sebrell, W. H., and Cleckley, H. M. Publ. Health Rep., Washington, 1940, v. 55, p. 157
Lancet, 1939, v. 2, p. 1178.
———, 1941, Annotation, "Cow's milk for neonatal haemorrhage," 1941, v. 2, p. 250.
Langdon, H. M. Amer. Jour. Ophth., 1928, v. 2, p. 386.
Manson-Bahr, P. Lancet, 1940, v. 2, p. 356.
Métivier, V. M. Trinidad & Tobago, Council Paper No. 74, 1932.
———. (a) Lancet, 1937, v. 1, p. 779.
———. (b) Brit. Jour. Vener. Dis., 1937, v. 13, p. 246.
———. Amer. Jour. Ophth., 1941, v. 24, Sept.
Moore, D. G. Fitzgerald. Ann. Trop. Med. Parasit., 1934, v. 28, p. 295.
———. Lancet, 1937, v. 1, p. 1225.
———. Jour. Trop. Med. (Hyg.), 1939, v. 42, p. 109.
———. (a) Nutritional disturbance and optic atrophy. Brit. Med. Jour., 1940, v. 2, p. 848.
———. (b) Jour. Trop. Med. (Hyg.) 1940, v. 43, p. 190.
Moore, J. E., and Woods, A. C. Amer. Jour. of Ophth. 1940, v. 23, p. 145.
Morgan, O. G. Trans. Ophth. Soc. United Kingdom, 1937, v. 57, p. 64.
Nutrition in The Colonial Empire, Economic Advisory Council, London, 1939.
Oden, J. W., Oden, Jr., L. H., and Sebrell, W. H. Publ. Health Rep., Washington, 1939, v. 54, p. 790.
Scott, H. H. Ann. Trop. Med. Parasit., 1918, v. 12, p. 109.
Sebrell, W. H., and Butler, R. E. Publ. Health Rep., Washington, 1939, v. 54, p. 2121.
Sharples, L. R. Jour. Trop. Med. (Hyg.), 1929, v. 32, p. 358.
Spies, T. D. Amer. Jour. Med. Sci., 1939, v. 198, p. 40.
———, Vilter, R. W., and Ashe, W. F. J. Jour. Amer. Med. Assoc., 1939, v. 113, p. 931.
Spies, T. D. Trans. and Studies, College of Physicians, Philadelphia, 1940, v. 8, p. 12.
St. John, C. H. Personal Communication, 1930.
———. Malnutrition in patients attending the eye department of Barbados General Hospital (privately printed) 1936.
Stannus, H. S. Trans. Soc. Trop. Med. Hyg., 1912, v. 5, p. 112.
———. Trop. Dis. Bull., 1936, v. 33, pp. 729, 815, 885.
Strachan, H. 1888, 1897. Quoted by Stannus, 1936.
Sydenstricker, V. P., Sebrell, W. H., Cleckley, H. M., and Kruse, H. D. Jour. Amer. Med. Assoc., 1940, v. 114, p. 2437.
Traquair, H. M. Private Communications, 1933, 1935.
Wille, W. A. Brit. Jour. Ophth., 1938, v. 22, p. 705.
Wright, E. J. 1927. Quoted by Wright (1934).
———. Dietetics in Warm Climates. Leitch, London, 1930.
———. Brit. Med. Jour., 1934, v. 2, p. 234.
———. Ibid., 1936, v. 2, p. 707.

CEPHALIC TETANUS FOLLOWING INJURY TO THE EYEBALL*

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Cephalic tetanus is a clinical form limited to the cephalic region, following an injury to the head, mainly to the orbito-naso-temporo-malar region, accompanied by unilateral or bilateral trismus, contraction of certain facial muscles and even of the muscles of the neck, and facial paralysis.

As a rule, the incubation period is long, more than one week, and trismus precedes the facial paralysis. The latter is the more serious the earlier it appears; unilateral, on the side of the injury, or bilateral in the case of injuries of the median line, giving to the face the classical aspect of paralysis on the affected side and contraction on the unaffected side.

Ordinarily, the condition begins with trismus, which is unilateral, spreading afterwards to the opposite side; however, with less intensity. The contraction of the maxillary muscle is followed by the contraction of other facial muscles, those of the nape of the neck and of the neck, involving first the injured side, afterwards creeping to the opposite side; then the facial paralysis occurs. The latter displaces the mouth, lowers the labial commissure, erases the labial line, causing the cheek to become flaccid, prevents the closing of the corresponding eyelid, and disturbs mastication as well as deglutition and the articulation of words. The association of the paralysis of one side with the contraction of the opposite side causes the strange aspect of risus sardonicus.

The facial paralysis, which ordinarily is localized on the side of the injury, presents the characteristics of peripheral facial paralysis, not affecting the motor nerves of the auditory ossicles, those of the soft palate and uvula, or of the chorda tympani, so that there is no disturbance of the sense either of hearing or of taste. This peripheral paralysis may be complete, superior and inferior simultaneously, or incomplete. In such case there may be either an inferior type limited to the inferior branches of the lingual triangle, or to the median branches; or a superior type with lagophthalmos, removal of the frontal rugae and integration of the muscles of the nares, of the lips, and of the jaw; or a total paralysis, involving the intrapetrous nerve branches and causing hyperacusia and disturbances of the palate limited to the paralyzed side, as was observed by Lortat-Jacobs in 1902.

The facial paralysis generally disappears with the trismus, preceding it, as I observed in my cases, or following it; it may even remain after the tetanus has been cured. It is not the only paralysis observed in cephalic tetanus, for it may be associated with ophthalmoplegia. This form, called paralysis of the bulbus, was studied by Worms, after Wahl in 1882 had mentioned it, and it was given the name of tetanic ophthalmoplegia. It usually follows an injury to the orbito-superciliary region, contusion or fracture of the superciliary region, an injury to the eyelid, or injury to the eyeball, as was the case in one of the patients observed by me. The injury may be caused by a bullet, by the lash of a whip, as in the case of our first patient, or by a stone, as in our second case.

Zack saw this form of tetanus appear

* Presented at the first Pan-American Congress of Ophthalmology, in Cleveland, Ohio, in October, 1940.

following an injury to a foot, and Shalier and Glenard also cited some examples of exceptions to the general rule.

In rare cases, as was witnessed by Lepine and Sarvonat, ophthalmoplegia preceded the trismus by a few days. The ophthalmoplegia manifests itself by ptosis and immobility of the eyeball, the external rectus as a rule being less affected than are the other oculomotor muscles.

The ptosis, which is the chief manifestion of the paralysis, may be: unilateral, a fact verified in our second case and previously mentioned by Hadlick and Solmsen and by Harnani Alves; or bilateral, as cited by Rockliffe. In the beginning the ophthalmoplegia may be total or localized; it may be progressive and bilateral, as in the case of Roberts and Williamson; it may also involve only the external musculature of the eye, leaving the internal intact, as in the case of Marx.

Wahl cited paralysis of the two internal recti. Isolated paralysis of the sixth pair is rare; that of the fourth is exceptional. When the internal musculature is affected miosis and loss of reaction of the pupil to light are noted.

Lepine and Sarvonat have stated that when the pupil is contracted, it may be dilated by atropine. Mydriasis was observed by Jacobsen.

In the earlier literature Larrey and Harkness mention amblyopia.

Exceptionally, a paralysis of the hypoglossal nerve is associated with the facial paralysis and with ophthalmoplegia; in which case may be observed the syndrome of labioglossolaryngeal paralysis.

Cephalic tetanus may remain purely spasmodic, without the occurrence of any paralysis. This form, called simple, presents unilateral or bilateral trismus in addition to contractions of some of the facial muscles and even cervical opisthotonus of divers degrees, as well as a pharyngeal spasm that disturbs deglutition, the dysphagial form. In rare cases the pharyngeal spasm attains such an intensity that phenomena of hydrophobia result, provoked by a touch of the pharynx, by deglutition, or even by the mere sight of liquid, the hydrophobic form. Sometimes there are also spasms of the diaphragm and of the respiratory muscles, causing dyspnea and asphyxia. Finally, cephalic tetanus is essentially characterized by contractions limited to to the face and the neck, associated with a facial paralysis more or less extensive and, less commonly, with ocular paralysis. Ordinarily it is apyretic or accompanied with but little fever, and is of relatively slow progress, as was true of my cases.

Historical

Cephalic tetanus was first described by Edmond Rose of Zürich, in 1872, in the Encyclopedia of Pitha and Billroth, to which he gave the name "Kopftetanus" or cephalic tetanus, having observed two cases. Following him, several investigators undertook the study of this clinical form, and there appeared the observations of Pollock, Huguier, and Langenbeck, the last-named having been the first to use chloral as therapy. After these came Kirchner with an observation, and in 1879 the case reported by Zigmondy and Kirchoff. Themée in 1880 presented an observation of interest in that a median injury with facial diaplegia and bilateral trismus was described. In the same year Gosselin published a case of unilateral tetanus simulating, at first, a facial paralysis. In 1882 appeared the first anatomopathologic researches of Von Wahl and Lehrnbecher; in 1883 the observations of Middeldorff and Meyer, Nankivell and Bond. Bernhardt in 1884 referred to cases presented before the Medical Society of Berlin, stressing the seat of the lesion. In the same year Guter-

brock observed a case, giving great importance to cold as a factor in inducing facial paralysis.

The cases of Triglia, Wagner, and Hadlick were presented in 1885 and in the next year those of Oliva and Brunner. Sereins also published a case with palpebral ptosis in that year. In 1887 Giuffré, Grossouard, Maysurins, Dumalard, and Terrillon published new cases. The latter and Schwartz, in a memoir published in the review of surgery in 1888, described an observation in which they outlined the principal characteristics of cephalic tetanus. In that year cases were observed by Renny, Charvot, Phelpes, Buisson, and Perret. Willard (Gaz. des Hop.) published a critical memoir presenting various observations. In 1889 Klemm and Lannois described several cases. Albert, of Lyon, in 1890 published a thesis presenting the observations of Perret and Lannois. Campos, of Havana, in the same year cited a case of ocular paralysis, and Rockliffe described another with bilateral ptosis. In 1891 there appeared the observations of Williamson and Roberts (in the Lancet), those of Von Spange of Holland, those of Otto Brennecke and Rudolf Behr.

Two personal observations were published in a paper by Jannin in 1892, in which he divided tetanus according to the predominant localization of the toxin in the nerve centers: medullary tetanus or generalized, and bulbar or cephalic tetanus. Max and Caird in the next year described certain cases, and in 1894 Houques's thesis appeared. He studied cephalic tetanus in association with various paralyses. Fromaget's publication in that year containing observations of several cases appeared in the Archives d'Ophtalmologie.

In 1895 Schnizler's histological studies appeared, following an autopsy in a case of cephalic tetanus, in which he verified nuclear lesions, and in 1896 Le Dard's detailed study of facial paralysis in that form of tetanus.

In the year 1889 Wellner reported a typical case of facial paralysis associated with tetanus, in the Revue de Neurologie, based on autopsies and giving the entire history of this problem to date. He disagreed with Brunner, who denied the existence of true paralysis in cephalic tetanus. The observations of Nothnagel and Joly were reported in 1901-1902, and those of Haltenhoff, interesting for the ocular paralysis cited. Worms, in 1904, studied the ocular paralysis in this form of tetanus, calling it paralytic bulbus, and in the same year there appeared the thesis of Poan de Sapincourt, who discussed cephalic tetanus with facial paralysis, presenting observations from various authors. Achard, in 1923, in his book entitled "Clinique médicale de l'Hopital Beaujon" described this form of tetanus in detail.

In Brazil, Jose Pereira Guimarães described a case in 1893, Hernani Alves two cases in 1910. Since then, there have been only the cases described by me, the first in 1920, the second in 1939. However, it should be emphasized, that my cases are the only ones in which cephalic tetanus followed injury to the globe or adnexa—eyelid and globe in the first case, ocular globe only in the second. In all others the origin of the injury was localized elsewhere in the body.

Passing now to the citation of cases whose origin was an injury to the ocular globe itself (exceptional cases in that only those to be mentioned in the following paragraphs were found in the literature) it should be stated that there may have been other cases of this kind not reported, and it is therefore probable that the occurrence is not so rare as it would seem from the paucity of reports.

Chappé referred to cases of cephalic

tetanus caused by ocular injuries which were generally benign. Jacqueau advised preventive injections of antitetanic serum in case of ocular injuries.

Genet advised the application of the same serum as a preventive in cases of ocular injuries occurring in farmers and masons.

Castellain and Lafargue mentioned a case of cephalic tetanus following an ocular injury caused by iron, which was cured in 45 days after intravenous injection of antitetanic serum in large doses, cleansing the area of injury with peroxide and subcutaneous injections of carbolic acid, to which he attributed great value in combating the contractions. In his 24 cases of cephalic tetanus of ocular origin, four were cured and 18 of the patients died.

Van der Hoeve reported two cases of tetanus of ocular origin, in the first of which the disease manifested itself on the seventh day after injury. It was localized in the superciliary region, where were found fragments of wood mixed with spores of tetanus. He used the Baccelli method of treatment, but the patient died on the 16th day. In the second case the injury was to the conjunctiva and eyelid, the period of incubation being 11 days. Therapy consisted in the use of antitetanic serum, sulphate of magnesium, and chloral hydrate, the patient recovering. In the first case there was facial paralysis and of the abducens, in the second paralysis of the abducens and of the oculomotor nerve.

Addario la Ferla reported two cases of tetanus of ocular origin: the first that of a corneal injury followed by ulcer and hypopyon and by a homolateral facial lesion. He used the serum, but in spite of therapy, paralysis of the oculomotor nerve ensued on the opposite side, disturbances of deglutition, then death on the 11th day. In the second case the injury penetrated the cornea and was followed by loss of the eye. The first symptom of tetanus appeared three days after the injury, and was successfully relieved by the serum.

Bonnet, Paufique, and Perron published the description of a case in a 7-year-old child in which the injury was localized in the right lower eyelid; the first symptoms of tetanus appeared on the ninth day. In this case, as in my second case, there was conjugate paralysis of the facial and of the oculomotor. Serum treatment brought about a cure.

Maurice Gauthier and J. Rouvier reported a case of tetanus of nonocular origin in which they attribute a paralysis of the III cranial nerves to the antitetanic serum used. I do not agree with this view, but believe the paralysis due to the tetanic toxin, not to the serum, the paralysis having manifested itself tardily, as is sometimes the case in paralysis of the VII cranial nerve.

Jebay in 1937 presented a case in which an injury of the right upper eyelid was followed by paralysis of the levator palpebrae muscle, superior and inferior recti and internal rectus on the same side, in addition to paralyses of the internal rectus and of the left inferior oblique. After treatment with intravenous injections of serum there was regression of the paralyses in the inverse order of their appearance. The author attributes the lesions to the action of the toxin in a centripetal direction along the motor nerves.

Groenouw called attention to the rarity of cephalic tetanus of ocular origin, citing nine instances, excluding, however, that of Genth, in which in addition to ocular injuries there were lesions in various parts of the face. These were cases described by Pollock, Schultye and Ramiro Guedes of Lisbon, in which the origin of ocular injury was a blow with the lash of a whip, as in our case 1; that of Fro-

maget was caused by a skyrocket, and that of Kirchner by a bullet, whereas that of Chevalier followed cataract extraction.

Rust declared that the injuries were almost always of the penetrating variety, or small in size. The case reported by Samelsohn was one of only conjunctival injury. In several cases there was iritis, hypopyon, and panophthalmia; in one, sympathetic ophthalmia. Of these nine cases, only two ended in cures, after three to four weeks of therapy; the patients in the others having died within two or three weeks.

The case of Mauch, which was fatal, had a corneal origin—traumatism from a small bit of dirt. E. R. Schneider of Copenhagen cited 19 cases which he treated with serum, successfully. In the 22 cases cited by Castellain and Lafargue, 9 were included which had been reported by Groenouw.

Etiology and Bacteriology

Cephalic tetanus is a rare disease, as compared with other forms of tetanus. The general etiology is that of common tetanus. Cephalic tetanus follows in every case an injury infected with the bacillus of Nicolaier, accompanied or unaccompanied by other bacilli that might increase its virulence. The forms of cephalic tetanus called "a frigore" are no longer accepted, nor can there be admitted as tetanus the varieties of facial spasm described by Colles and Follin, following injuries to the face or skull. The special conditions necessary to the development of this form of tetanus are not like those of common tetanus: whereas this one is extremely rare following injuries to the face, cephalic tetanus always follows such injuries, whether internal or external, localized in the area of the superior facial nerve, mainly at the rim of the orbit, superciliary arch, root of the nose, inferior region of the frontal sinus, eyelids, and ocular globe. These comprise the pathognomonic characteristics of the disease.

Rare are the cases of cephalic tetanus following an injury to the orbit, to the lids, and mainly to the eyeball; however, cases observed following such injuries are more obvious. It is also rare to observe ophthalmoplegia preceding trismus, as in the case cited by Lepine and Sarvonat.

As to age and sex, this is as in common tetanus. Men are more exposed to infection for occupational reasons than are women. It is especially in youth and adult age that this form of tetanus is found, nevertheless it has been observed at all ages, from four months (Hadlich) to 69 years (Lannois) and 76 years (Bourgeois). There are opinions as to the influence of the seasons. Some investigators hold cold and the rainy season to be important factors in the rapid development of the infection. Occupation also is a factor to be considered; the disease is more likely to be found in coachmen, blacksmiths, troopers, gardeners—in short, in those who come in contact with the soil.

Bacteriology. If the chapter on pathologic anatomy is still obscure, that on bacteriology is, on the contrary, one of the best known. The first bacteriologic researches were made by Remy and Villar, and were negative; as were also those by Perret, Roux, and Lannois. Rappin, however, had a positive result, the inoculation of two guinea pigs with a fragment of the foreign body proving fatal; one by Vibrio septicus, the other by Clostridium tetani, the latter with trismus at the moment of death.

The research of Lortat-Jacob was still more positive and interesting. A guinea pig inoculated in the left inguinal region died in left pleurothotonus, after presenting as early symptoms contractions localized on the side of the inoculation; that

is, on the left side. This is one of the most instructive facts in regard to the tendency of tetanic toxin to localize itself in the vicinity of the inoculation.

In 1890 Knud Faber showed that Cl. tetani when cultivated in a liquid medium secretes a soluble toxin that persists after filtration, and when injected into animals reproduces the typical picture of tetanus. This author observed that the injection of the toxin is not immediately followed by contractions, as is observed in the case of strychnine, but after a period of incubation. The ingestion of the toxin is harmless; the intravenous injection determines the generalized tetanus. Knud Faber considers tetanic toxin to be a toxalbumin akin to the virus of rabies, which is destroyed in five minutes at a temperature of 65°C. In fact it is very attenuated at that temperature.

Pathogenesis

In regard to pathogenesis, there are serious difficulties in explaining how tetanus, an extraordinarily convulsive infection, should simultaneously present paralytic phenomena and contractions. Various theories have been proposed. Rose proffered two theories: one asthenic, in which the paralyses were considered similar to the muscular relaxation of death throes in acute diseases; however, its precocious character and its appearance in some cases on the eve of death made him abandon it and seek another explanation, which he called the mechanical. Referring to the facial nerve, he said that due to the contact of its terminal fibers with the tetanic toxin, there was an irritation that followed the agglutination of the nerve, hence its strangulation at the foramen of the mastoid process, which becomes too small to contain it. The seat of that lesion explains the absence of disturbances toward the side of the ear and the palate. Autopsies did not confirm the hypothesis of Rose, since there was no evidence of strangulation nor of nerve compression.

The reflex theory proposed by Sereins, in 1880, admitted that the irritation of a sensory nerve such as the trigeminus, stimulated the nerve centers to the point of determining paralysis of a motor nerve of the same innervation area. Houques shared Sereins's opinion, explaining facial paralysis by the influence of the trigeminus upon it. Gouwers also accepted it and stated that stimulation of the trigeminus, produced at the moment of avulsion of a tooth, may cause a paralytic ptosis. Objections were raised to Sereins's theory, Zack's case being pointed out in which a paralysis of the IV nerve manifested itself following injury of a foot. How is the persistence of the motor reaction to be understood when there was no longer sensibility? Because the sensitive trigeminus is not reflected upon the motor and goes through paths of anastomosis to reflect itself upon other nerves.

The infection theory was accepted by Olivier, who attributed to facial paralysis an infectious origin. Perret sustained it and Lannois admitted it. Albert (1890) had the same conviction, believing the products isolated by Brieger to be the cause of convulsions in various degrees. One of these products—tetanus toxin—weakly convulsive, is translated into a generalized muscular paralysis. Brunner in 1894 succeeded in reproducing a hemicontraction and paralysis of part of the snout of a guinea pig, on the side into which he had injected the toxin, attributing paralysis to a contraction produced by a heightened virulence or to the prolonged action of the toxin, concluding therefrom analogies with the toxin of rabies, which causes paralytic phenomena when applied in large doses, and convulsive phenomena when used in small doses.

Clinical observations seem to corroborate Brunner's opinion, inasmuch as cephalic tetanus, accompanied by facial paralysis, is generally serious, and the hypothesis may be admitted that there is a great absorption of the tetanus toxin by the system. The anatomic disposition of the region seems to explain the predilection of the tetanus toxin for the facial nerve. In that region there is a vast and superficial nerve plexus, whence the lesions of its ramifications by the tetanus toxin. There is also neuritis, which may be attributed to the vulnerability of the facial nerve, and verified by the frequency of paralysis *a frigore*, or again by the sensitivity of the cells if its nucleus of origin, is more sensitive to the action of tetanus toxin.

Albert believes that in the face the action of the toxin lasts longer because the anatomic planes are opposed to its rapid diffusion.

In the opinion of Brunner the toxin at first causes a rapid and brief contraction of the muscles, followed immediately by a secondary paralysis of the same muscles. The action of the toxin depends upon its virulence and upon the conditions of absorption peculiar to the region; as the facial nerve forms in the face a plexus of numerous and superficial networks, and as the substance has great affinity for tetanus toxin, it is absorbed. The result is a disturbance of its motor function and a facial hemiparalysis. There is, then, a local action of the toxin upon the facial nerve, contrary to what is found in the motor branch of the trigeminus, which is reached only through the circulatory system, as in ordinary tetanus; the result is a paradoxical combination of a contraction with paralysis.

As for the ophthalmoplegia that has been observed in several cases, it is true that authors have let it pass unnoticed, notwithstanding its existence in cases of injuries localized in the orbital area, in the eyelids, and in the globe itself.

We may admit that in this case the ocular nerves or their nuclei of origin are less affected by the virus of tetanus.

Worms accepted Brunner's concept relative to the action of the toxin of tetanus, that it was according to the virulence of the toxin, admitting, however, that the cranial nerves react in a different way in relation to the virus. He also admitted a nuclear origin for the paralyses and concluded that the greater or lesser affinity for the toxin resides in the ganglion cells of the affected nerves.

Also interesting is the confirmation of the origin of the paralyses. Various investigations have been undertaken to solve this problem. Rose, maintaining the peripheral origin of the facial paralysis, invoked the phenomena of compression in the aqueduct of Fallopius and the mastoid process, whence the functional disturbance. As is known, he found no lesion in the facial nerve (intracranial, petrous, and peripheral trajectory).

Roberts, Schnitzler, Caird, and Schupfer verified nothing upon examining the motor nerves. The changes in the cerebral hemispheres, much diffused, were not sufficient to explain a total paralysis of the facial nerve nor of the ocular nerves unaccompanied by paralysis of the extremities and of the tongue. Mental disturbances were never observed. The nuclear changes were of great importance after the appearance of modern methods of staining, and have held the attention of investigators. Bernhardt, having found no lesions in the tests made on the peripheral nerves, assigned to tetanus paralysis of the cranial nerves a central origin, and under the name of vacuolar aspect, described modifications in the nucleus of the paralyzed nerves (Elisher).

In 1892 these numerous changes were observed by Merlich in the ganglion cells

of the facial nerve, trigeminus, oculomotor, and hypoglossal nerves. Schupfer and Radlich also found changes of various intensity in the several bulbar nuclei.

Probagensky and Lortat-Jacob verified the changes on the side of the paralysis. How should be explained the ocular paralyses observed on the opposite side to the injury? What explanation is there for the case of Schupfer, in which there was an associated paralysis of the eyeballs? We believe they may be likened to the postdiphtheric paralyses, which by their multiplicity and extension, and the participation of the cranial nerves and of the respiratory centers seem to depend on the same origin, although some authors, particularly Bambouneix, admit an ascending neuritic origin for these paralyses.

The nerve branches thus present themselves as endowed with a special conductivity, incapable, however, of being changed by the action of the toxin; that is, without inflammatory phenomena or neuritis. They are veritable channels through which the tetanus virus reaches the central neurons.

The localization of the paralytic phenomena in the vicinity of the point of inoculation explains the attack of the toxin being limited to the nuclei of origin of the motor nerves in the region.

There is, however, the case of Zack, in which an individual after being injured in the foot presented paralysis of the patheticus four weeks after the accident. For an explanation of this, we need only admit that the toxin, diffusing through the peripheral conductors, spreads through the entire central nervous axis, reaching only the bulbar cells of the nucleus of the patheticus.

Does the tetanus toxin affect in the same way the sensitive motor fibers? In analyzing this question, we know whether the virus of tetanus is conducted to the centers by the motor branches of the facial and of the oculomotor, or by those of the trigeminus (Willes's branches). Marie and Morax, upon the basis of their cases, believe that the sensory fibers absorb the toxin in lesser quantity than do those of the motor. Others, such as Meyer and Ranson, say that the motor nerves are better conductors, but that the propagation of the virus may also take place by means of the sensory fibers. Courmont, Doyon, and Autokratof believe that the motor and the sensory nerves serve equally well as regards conductivity. In view of these conclusions, we may admit that tetanic paralysis of the cranial nerves may have two origins; one through the trigeminus, the other through the facial nerves, oculomotor, and so on.

As to the paralysis of the seventh pair, it is certain that tetanic toxin may be conducted to the superior or inferior facial, according to the localization of the injury in the region of the one or the other.

In regard to ophthalmoplegia, whether it be admitted with Duval that the fibers of the superior facial assigned to the frontal muscles and to the superciliary and orbicularis muscles derive from the sixth pair, or, as stated by Mendel, they come from the third, it is explained not only by the paralysis of the facial but also of the oculomotor and the external oculomotor. Duval's hypothesis has few adherents; that of Mendel is upheld by some and contested by others, chiefly Marinesco.

Worms in an attempt to explain the fact states that in the majority of cases in which there was ophthalmoplegia, the lesion was situated in the region of enervation of the oculomotor, and for this reason the toxin reached the bulbus through that nerve, inasmuch as its paralysis seldom fails whenever the injury is localized in its territory of enervation, whence the virus may be carried to the

opposite side. This explanation is acceptable inasmuch as it explains the means of propagation of the toxin to the bulbar centers. The tests seem to demonstrate that the propagation of various cranial pairs whose zone of distribution is outside the injury is effected exclusively through a centripetal pathway and results from transmission of the toxin to the bulbus. Achard in histological examinations on mixed nerves of the regions near tetanus-producing injuries, found, in cases of fatal generalized tetanus, a small number of nerve branches showing lesions and descending degeneration, admitting that the trophic centers were injured, small lesions being found in the nerve centers; and Nageotte Ettlinge and Manouelian described changes in the nerve cells, which, however, do not seem to explain the contractions, as Buck and Demoor stated. This would explain, however, the possibility of the paralysis, but it would remain to determine how these paralyses are present only in cephalic tetanus. Perhaps they result from a modification of the nerve centers which prevent them from reacting as usual to the action of the toxin.

In summary, it should be said that the pathogenicity of cephalic tetanus is still far from being completely explained, requiring further research.

Pathologic Anatomy

Most fantastic lesions have been described in autopsy reports on patients who have died from tetanus.

Larrey mentioned inflammatory lesions of the medulla with a flow of reddish serum and congestion of the peripheral nerves; also congestion of the brain. The meninges were observed to be hyperemic, with a bloody exudate and subarachnoid serous infiltration.

Charcot and Michaud described a central superacute myelitis. Microscopically, Bouchard saw proliferation of the neuroglia, pigmentation of the cells of the anterior horns and of the adventitia of the capillaries of the medulla.

Arloing and Tripier insist upon hyperemia and medullary proliferation. Broca mentioned general congestion of the nerve axis and softening in the cervical or lumbar areas. Many other lesions were described by numerous authors, but the anatomo-pathologists did not verify them in the cases they studied.

Robin did not find lesions in the medulla nor in the brain. Ranvier, in four observations, did not report any lesions, and the same is true of Hayem and Vulpian. Marinesco observed fine lesions in the cells of the anterior horns of the medulla, produced by tetanic toxin. A part of the cell in the vicinity of the cylinder axis was opaque or without histologic structure; the cylinder axis became granular, staining with blue by Nissl's method, whereas normally it is colorless and of homogeneous appearance, the protoplasmic prolongations having the aspect of knotted clusters irregular in contour without apparent trace of chromatophyl elements. Rispal found similar lesions in the medulla of tetanus victims; verifying in one case tumefaction of the cellular body of the nucleolus and of the corpuscles of Nissl, with chromatolysis more or less accentuated. In another case he noted a hyaline transformation of the protoplasm with deformity and displacing of the nucleolus. Many other authors, along with Nissl, Goldschneider and Flateau, Pechoutre, and others, found cellular lesions. All of these histologic changes are considered by the several authors to be specific of the toxin or determined by the high fever, contractions, spasms, and other symptoms.

Courmont, Doyon, and Paviot consider these lesions inconstant and when present to lack specificity. Nageotti and Ettlinger

recognize the existence of cellular lesions, but do not consider them the cause of typical symptoms.

Examination of the peripheral nerves did not furnish positive data. In addition to those lesions were mentioned interstitial hemorrhages, degeneration, and chemical changes in the muscles.

It should be noted that the lesions described in the nerve axis and in the muscles are out of proportion to the violent manifestations of tetanus: hyperthermia, contractions, recurrent spasms, and so forth, which appear as a consequence and not as the cause of tetanic states.

Examination of the cerebrospinal fluid, taken during the course of the tetanus by Sainton and Maille, produced negative results; the fluid was sterile and presented no leucocyte reaction, nor did it contain albumen. Inoculation into guinea pigs induced neither toxic nor infectious conditions. There was found in the cerebrospinal fluid an appreciable quantity of glucose, which seemed to decrease in the cases which had become aggravated.

Earlier authors who were interested in the anatomo-pathologic researches thought they had found the explanation of the mechanism of the clinical symptoms; the more recent investigators' hope was in the study of the bacteriology —in their opinion it explained all the symptomatology of that affection.

We know almost nothing of how tetanic toxin interferes with the nervous system. The microscope reveals nothing regarding the modifications caused by the toxin in certain cells, nor as to which cells they are; the investigators attempted to fill this void by making a kind of physiologic dissection, with which they might be able to determine the functioning of the reflexive arcs in the course of tetanic infection. Their reports and deductions are instructive but could not deserve the confirmation of histologic proofs, which are beyond discussion. This chapter, which in the study of tetanus occupied a prominent place before the era of bacteriology, has almost disappeared from modern treatises.

Diagnosis

The diagnosis of cephalic tetanus is relatively simple because of the characteristic contractions and paralyses, as well as of the existence of trismus and of an injury in the vicinity, which factors lead one immediately to think of tetanus. There are, however, circumstances that may make the diagnosis difficult; such as, for instance, the existence of a healed sore not mentioned by the patient when he is examined, or when the facial paralysis precedes the trismus—a fact that may lead one to think of a traumatic facial paralysis.

A traumatic lesion of the small deep petrosal would be accompanied by signs peculiar to the localization, rare in cephalic tetanus. The unilateral trismus of tetanus is very easily distinguished from painful tic of the face and from hysterical facial spasm, the characteristics of which are different in the circumstances referring to their appearance and in their clinical manifestations as well. The ophthalmoplegia of cephalic tetanus should not be confused with a contraction which might be localized in the opposite side. The perimetric exploration of the movements of the ocular globe would prevent this error; besides, in the spasms are observed involuntary winking, fibrillary contractions of the palpebral orbicularis, and sometimes nystagmus as well as rapid movements and the inconstancy of the contractions, unlike the permanency of paralytic disturbances.

Once the ophthalmoplegia is recognized it becomes necessary to eliminate other possible causes of ocular paralyses. Those that are caused by an acute meningitis, such as the cerebrospinal, are preceded by general prodromes of the disease and

with fever; they are accompanied by psychic disturbances, sign of Kernig, and not by true trismus, lumbar puncture removing all doubts.

In tuberculous meningitis of tetanic form, described by Boix, may be observed trismus, rigidity of the nape of the neck, and ocular paralyses; but there are no spasmodic paroxysms as in tetanus; on the contrary, there are other symptoms peculiar to meningitis, and lumbar puncture will reveal meningeal reaction.

A traumatic ophthalmoplegia results most frequently from a fracture of the orbit with intraorbital hemorrhage, while cephalic tetanus usually results from a light and superficial injury. Fracture of the base of the skull causing an ophthalmoplegia would be accompanied by other symptoms, such as divers paralyses and coma; there would be no trismus, a fact of great importance because of the period of incubation which characterizes cephalic tetanus.

In our observations there was no difficulty in making the diagnosis, inasmuch as the existence of an injury (sore), of the clearest trismus, and a facial paralysis, and in one of them blepharoptosis, left no doubt as to diagnosis. The incubation period was of 11 days in one case and of 12 days in the other, and the blepharoptosis was on the same side as the injury.

Progress

As in common tetanus, the cephalic form follows an acute phase, a subacute, and a chronic. When the acute phase is in progress the prognosis is always worse; if the evolution is slow it is more favorable, the disease terminating more frequently in a cure, as in my cases. The duration of this affection varies, depending on the progress followed by the disease. Cases have been reported in which the duration did not exceed two days, ending in death; others in which there was a course of five to ten days; and still others of much longer duration, lasting more than four weeks and ending favorably, as we observed.

Prognosis

The prognosis of cephalic tetanus, like that of the localized form, in general, is relatively good, or at least less serious than in common tetanus. Schneider of Copenhagen, however, in 19 cases of ocular tetanus, reported only three cures, and Castellain and Lafargue only four in 22 cases.

The prognosis must be reserved, although it is less grave than in common tetanus. Some factors aggravate it; for instance the progress of the disease, the duration of the incubation period, the early generalization and the degree of toxicity, as well as the age and resistance of the individual; also the early diagnosis, for on it depends the application of a rapid and prompt treatment, which will greatly influence the evolution of the disease.

If the number of injuries increases the gravity of the infection, multiplying the foci where the toxin is produced, the focus seems to be of less importance in relation to the prognosis.

Fever is considered important for the prognosis. However, there are cases in which fever was present yet which ended in cure, and inversely apyretic forms resulted in death.

Bernard and Lumière think that the existence of contractions of the abdominal muscles is an advance signal of a contraction of the respiratory muscles, bringing on asphyxia. However, in partial tetanus, there is frequently abdominal contraction, and the prognosis is generally favorable.

Treatment

Treatment of cephalic tetanus, as in all other localized forms of tetanus, does not differ from that of common tetanus. The therapy has in view the modifying of the

condition of the injuries and to rid them of the several microörganisms that they may contain, combat the contractions and spasms that threaten the muscles necessary to life (vital muscles), and neutralize the toxin. The lesions should be carefully cleansed of foreign bodies, coagulation of blood, and lacerated and necrotic tissues. Injured limbs have been amputated, but such operations have not protected the patient from tetanus. Antiseptic cleansing fluids should be made up of several germicides in the pharmacopeia: oxygen in the original state, peroxide, persulphate of sodium (Lumière), phenic acid, Dakin's fluid in 5-percent solution, sublimate, mercuric oxycyanide, hot air, and the like.

If the injury is to the eyeball, milder antiseptics must be used. These irrigations do not destroy the spores of tetanus but may destroy germs, associated with them, whose role is of extreme importance. Surgeons in the war of 1914 insisted upon these prescriptions and established the fact that antitetanic serum, even when injected immediately, does not avert tetanus.

Dupuytren, Hervieux, and Renzi insisted upon serenity and the absence of excitement as necessary to the symptomatic treatment of tetanus. Renzi, 50 years ago, advised placing the patient in a dark room and closing the external auditory canal with wax. Opium was given by Larrey in a dosage of 0.25 to 4.0 grains in 24 hours. The latter figure was probably exceeded, reaching 7.0 to 8.0 grains of the extract per day. Morphine injections were advised as well as belladonna. Chloroform inhalations were employed by Eschallier, an intern of Velpau in 1847, and inhalations of amyl nitrite by Richardson in 1864. During the war of 1914 such inhalations were again employed by Martin and Darré.

Langenbeck in Germany and Verneuil in France recommended the use of chloral, which is still very much in use today. Chloral induces sleep, diminishes the contractions, abolishes the pain, and calms the excitability of the medulla. The dosage advised, 6 to 16 gr. per day, became classic and may be continued for days and even weeks. The usual routes of administration are by mouth and by rectum. In order to avoid intolerance it is necessary that the chloral be diluted (1:50) for administration by mouth or parenterally, the dosage not exceeding 3.0 gr. Subcutaneous and intravenous injections (Oré, 1872) constitute exceptional methods, inasmuch as the former provoke necrosis and the latter require extreme care since they expose to injury. The bromides are inferior to chloral. Curare appears to lead to occasional injury. Baccelli's method won numerous adherents but later failures relegated it to secondary choice. He recommended phenic acid, 2 to 3 percent in water in subcutaneous injections, more as a moderating measure for medullary sensitivity than as an antitoxic agent. According to Baccelli, the doses should be increased on an average of 1.0 percent per 24 hours, in several applications. It has been a method employed by me, in association at times with sulphate of magnesium in 20-percent solution, at other times with electrargol in intravenous injections, together with the parenteral injection of a 5-percent solution of bicarbonate of soda and prolonged tepid baths. Collargol has been employed, using the same route, with success.

Electrargol brought such relief to a patient of mine that he immediately asked for another injection. Magnesium sulphate diminished the contractions (Meltzer and Auer). The solutions of this salt are injected subcutaneously, into the muscles or into the veins or into the cerebrospinal fluid (Ramond, Griffon and Liau, and Tanton). Magnesium sulphate injected into the lumbar region has a more

constant and lasting effect, but this method acts only upon the contractions and has no curative effect. According to Jean Camus, this salt momentarily decreases the contractions and may provoke serious injuries when injected into the cerebrospinal fluid.

During the war Lumière and Bagy employed sodium persulphate. The former reported that the alkaline persulphates are capable of destroying tetanic toxin in man. This method consists in the injection into the vein of 20 c.c. of a 5-percent solution of pure and neutral sodium persulphate. These injections may be repeated once or twice a day for 8 to 15 days.

Leger and Walther recommend in some cases subcutaneous injections of oxygen, injecting one to two liters of this gas a day.

Specific therapy

The specific agent against the toxin is the antitoxin. It tends to produce the antitoxin in the diseased organism, active immunization; or where a prepared antitoxin is injected into the organism, passive immunization.

Active immunization. Henri Vallée and Louis Bazy tried antitetanic vaccination by mixing the toxin with an iodized solution, and injected into the patients a 4-c.c. dose which was increased to 12 c.c. This attempt was made because of the investigations of Roux and Vaillard. Vaccination with attenuated tetanic toxins, however, requires, before the animal acquires a sufficient immunity, a series of successive inoculations at long intervals, which makes it impossible as a treatment in so rapidly developing a disease as tetanus.

Passive immunization. The application of the specific antitetanic serum in man may be used as a preventive, before the manifestation of the disease, or for the purpose of curing it after the infection has set in.

Preventive Serotherapy: The employment of antitetanic serum in man as a preventive has raised many discussions. Long ago Roux and Vaillard proclaimed the beneficial results of this method. Various observers, such as Championnière, Bazy, Schwartz, Guinard, Vallas and others showed that serum therapy used as a preventive is of indisputable efficacy when applied to domestic animals, and no less so in man.

Reynier, based on an observation in which the serum when employed as a preventive did not avert the appearance of tetanus, long after the injection, denied the prophylactic value of the antitetanic serum. Vaillard then observed that in those cases the serum had been employed too late and in insufficient quantity; moreover, that the injections should have been repeated inasmuch as the antitoxin disappears rapidly from the organism. He believes that the main requirement for the efficacy of this method is that the injected antitoxin should remain in the organism long enough to act on the toxin. This has been fully demonstrated by the researches of Dehn and Hamburger. Vaillard also insisted that the serum is not bactericidal but antitoxic; it neutralizes the toxin wherever it exists, but does not prevent the tetanus bacillus and spores of the focus of infection from multiplying and producing more toxin. Because it is rapidly eliminated, the antitoxin must be injected repeatedly in order that it may act successfully. Vaillard advised injections of 10 c.c. of serum in the case of ordinary superficial sores and 20 to 30 c.c. in nonfracture injuries in which there is necrosis of tissue. The injected antitoxin persists for approximately one week in the human organism, but in order to decrease it afterwards it is necessary, two to six days later, to inject 10 c.c. before the end of the first week and even fortnightly, depending on the case.

Santos Fernandes in 1886 reported a case of tetanic infection after the excision of an intraorbital sarcoma, tetanus appearing five days postoperatively and death ensuing on the 10th day. He could not account for the infection.

Could the appearance of tetanus have been prevented in my patients? Yes, if the resources of the locality where the patients lived had permitted it and if it were admitted that, as a rule, every wound in direct contact with the earth, or dust, or objects contaminated by earth, which have been dragged on the ground or have had contact with horse manure is infected. Or if it is borne in mind that a superficial or deep injury caused by fragments of wood or metal, inasmuch as the tetanus bacillus is so widespread in such objects and its spores so resistant, is a source of danger and should be disinfected and serum used promptly.

Tetanus would have been prevented by prophylactic injection of Behring's serum applied as early as possible, within 30 hours after traumatism, in a dose of 20 to 40 thousand units, by subcutaneous or intramuscular injection in addition to thorough cleansing and disinfection of the injured part. If healing did not take place in a week, the injection should be repeated, 10 to 15 thousand units every week until there is final cicatrization.

Curative Serotherapy: As soon as the tetanus infection appears, curative serotherapy should be applied, as in the two cases described herewith, although this etiologic therapy is uncertain and inconstant. The serum should be employed promptly whenever possible, from the moment of appearance of the spasms and of the early warning symptoms, even before the appearance of trismus. The administrative means of introducing the serum are by subcutaneous intravenous, intraspinal and intracerebral injections.

Kitazato first applied the serum subcutaneously, but without success. Today it is currently employed, and it presents no technical difficulty, for which reason it is the preferred route. Large doses have been used. Gras injected 600 c.c., Le Noir and Michon, 630 c.c.; Sidney Long, 680 c.c.; Castaigne, Tourraine, and Françon, 760 c.c.; André Petit, 810 c.c.; Josué, Godlewsky, and Belloir, 880 c.c.; Castellain and Lafargue, 900 c.c.; Jean Selme and Tixier, 1,060 c.c.; D. Massary and Léchelle, 2,240 c.c.

Intravenous Injection was first employed by Morax, but without success, in 1892. This method has been recommended as the most rapid and efficacious, opinions being based on the researches of Arloing and of Nocard; however, they lead more frequently to anaphylactic injury. This method in association with the subcutaneous route seems to give the maximum probability of cure.

Intra-arterial, Intra-nervous, Para-nervous Injections: Heddans, later Gauthier and Chattot, injected antitetanic serum into the carotid, hoping by this means to carry the antitoxin more directly to the encephalic nerve centers. The serum was also injected into the large nerve trunks.

Sicard tried serum injections introduced in the vicinity of the nerves. Appert and L'hermite employed it in epidural injection in the sacral channel, intraspinally. The subarachnoidal injections had no results, 64 percent of the patients so treated died, according to Vaillard. Rollin, however, reported favorable statistics.

Hôtel injected 40 to 60 c.c. of the serum after lumbar puncture, and placed the patient in a declivous position, head down, to let the serum reach the bulbus.

Owing to the rigidity of the vertebral column it sometimes became necessary to use chloroform in order to carry out the lumbar puncture. This was tried and advised by Sicard in 1903. Recommended doses were 20 c.c., repeated on two or

three successive days, accompanied by hot baths, morphine, and chloral.

Intracerebral Route: Roux and Borrel proposed to make the antitetanic serum come in contact with the cells of the brain and of the bulb, by means of intracerebral injection, introduced through a narrow trepanation. Some successes were obtained, but it must be realized that if the toxin in the superior parts of the medulla has already taken effect, death will not be avoided, inasmuch as the antitoxin, even when carried to the brain, has only a preserving action and does not resolve the lesion already existent. This method, although interesting from the experimental point of view, is without advantage in man. The curative effect of the serum still remains inconstant. In our observations it did not produce the results hoped for, hence it was abandoned in favor of the use of carbolic acid associated sometimes with electrargol; at other times with magnesium sulphate together with warm (tepid) baths and parenteral injection of a solution of sodium bicarbonate, 5 percent, which brought excellent results.

Anaphylaxis: The exaggerated belief in anaphylactic injuries in serotherapy is prejudicial, since the injuries are not so frequent and means to combat them are available. Various methods of antianaphylaxis have been reported not only in relation to tetanus but also in regard to serotherapy in general. Paul Carnot, in an instructive report of the cure of an injured person after an anaphylactic shock, asked whether this shock could not have exercised a favorable effect on the evolution of tetanus. Jean Camus had attempted to utilize anaphylactic shock as an aid in experimental therapeusis. The results of his researches were inconstant and without practical application for man. A method seemed necessary that would not only carry the antitoxin rapidly to the level of the nerve centers but would also allow its penetration into the neurons. A shock analogous to that caused by anaphylaxis seems to fulfill these conditions.

What is the action mechanism of the enormous doses of antitetanic serum injected, which seem to have been so efficacious? In this therapy another fact should be presented in addition to the action of the antitoxin upon the toxin. If we remember that *in vitro* the smallest quantity of antitoxin is sufficient to neutralize doses of toxin that are often enormous, we may consider that the massive quantities employed have a vulnerable effect and toxic effect, by reason of which the action of the antitoxin is more penetrating, more intimate.

Conclusion. From what has been said, we may conclude that the treatment of tetanus should include antiseptic measures for the injury, consisting in excision, if possible, or cauterization with the thermocautery or tincture of iodine for the purpose of destroying the focus of infection. The cleansing and thorough disinfection of the injured part with peroxide, mercury oxycyanide, and such means remove the foreign bodies that are present; systemic injection of antitetanic serum in doses of 10 c.c., repeated from week to week, increasing to 20 c.c. in cases of anfractuous injury (sore, ulcer), using the subcutaneous route, the venous, or spinal whenever possible, in doses of 20 c.c.

For the alleviation of painful contractions, we should utilize narcotics such as morphine; hydrate of chloral, 3 to 4.0 drams in enemas preferably, repeated from 3 to 4 times each day or orally in strong doses, 12.0 and greater, in 24 hours.

In order to moderate the hypersensitivity of the nervous system, subcutaneous injection of carbolic acid should be used according to Bacelli's method.

In our patients, failing the serum, we

used carbolic acid in a solution of 3 percent mixed with sulphate of magnesium in a 20-percent solution, 10 c.c. per day; or electrargol, 10 c.c. twice a day in intravenous injections in addition to enemas of sodium bicarbonate, 5 percent, and rest, which brought great results.

Also employed were prolonged warm baths, milk diet, the patient being kept in semidarkness, silence, quiet.

The problem of the therapeusis of tetanus is one of the most disputed.

Tetanus is an affection whose cause we know as well as the pathogenic agent and the toxin, and against which we possess an antidote of indisputable efficacy; but when the toxin has fastened upon the nerve centers or has entered into its substance we are unable to effect its dissociation. From the results of Roux it may be deduced that the toxin may be dislodged from its combinations, particularly from its union with the antitoxin.

Experimentation and clinics have contributed to the collection of encouraging results; hence serotherapy which, up to this time, had been considered ineffective after the appearance of the first clinical symptoms, may furnish an appreciable amount of cures if well managed and accompanied by other therapeutic measure.

Case reports

Case 1. L. P. L., a Brazilian (brown) farmer, married, aged 46 years, was admitted to the Santa Casa de Misericordia on June 29, 1920. His parents were deceased, cause unknown; brothers living and well.

The patient had had measles in childhood, La grippe, and venereal infection. He admitted alcohol and tobacco intoxication.

On June 3, 1920, while driving a horse loaded with farm produce through places traversed by various animals, he whipped the horse, and the lash struck him in the left eye, traumatizing it severely, also injuring the eyelid. On the 15th of the month, 12 days after the accident, certain symptoms appeared. He had slept well, but was unable to open his mouth on awakening, felt pains in the left arm, which, however disappeared after three or four days. These pains came on in the morning and disappeared in the afternoon, to reappear the following morning. He was unable to swallow anything except water and milk, and these with difficulty, there being no reflux through the nose. His sleep was disturbed by suffocation and difficulty in breathing, which still occurs. The headache accompanying these symptoms lasted eight days.

Examination of face. Half of the face was immobilized and without expression. The frontal lines were erased on the left side and the lines on the right were only slightly visible; the supercilium was lowered. The patient opened his mouth more on the right than on the left side, the oral fissure being crooked, and the normal right labial commissure was drawn upward, the left naso-labial line erased. He felt no pain in the left temporo-maxillary articulation, and the labial movements were abolished on this side. There was, therefore, a facial paralysis of the left side, the same side on which the ocular lesion had occurred, and left unilateral trismus. When provoked to laughter the face assumed an expression of weeping, a painful smile, or the smile called sardonic or sarcastic.

Ocular examination. The bulb was atrophied as a consequence of the rupture of the cornea and of the sclerotic, the former being reduced to one-fourth its normal size. Upon touching the ciliary region the patient felt a slight pain. The upper lid presented an irregular cicatrix.

Other examinations. Gait, normal; sensitivity, normal; Ruine and Weber tests, negative; Romberg, negative; reflexes—superorbital, present on the right, abolished on the left; Masseter's, absent due to the trismus; plantars and abdominals, present; biceps, triceps, and patellar, present; Achilles, Babinsky and variants, absent. Temperature—this disease progressed without fever.

Evolution and treatment. On July 1st there was no change in the progress of the disease; the first injection of phenic acid was applied as a 3-percent solution in an amount of 3 c.c. The temperature remained normal, and there was no change in the pulse rate. On the 2d, the patient's condition remained the same. Another injection of carbolic acid was administered and two parenteral injections of 500 c.c. of a solution of sodium bicarbonate, 5-percent, were administered. From July 3d to 15th there was little change in the patient's condition. During this interval the injections of carbolic acid and the parenteral injections of sodium bicarbonate were continued, also prolonged warm baths. The patient was kept in semidarkness, in rest and quiet. From the 16th day onward an improvement was noted, inasmuch as the facial paralysis began to lose its original brightness until it disappeared altogether. The trismus also began to lose its clearness, but it still was observable, although but little pronounced, and more on the side of

the lesion, until the discharge of the patient on August 3d of that year. The treatment administered during that period was similar to that earlier in the course of the disease: injections of phenic acid, parenteral injections of bicarbonate of soda, and prolonged warm baths.

Baccelli's method, which was used in this case, gave excellent results. During the whole course of the disease there was neither fever nor alteration of the pulse rate.

Case 2. F. S., male, Brazilian (brown), single, aged 18 years, a domestic servant, was a resident of S. Jose do Tocantins, in the State of Goiaz. His family and personal history was without importance. On August 12, 1939, while playing with another boy, he was struck full in the left eye with a stone from a sling. He experienced great pain, and a sticky fluid flowed down his cheek. He washed the eye with cold water and afterwards, feeling complete loss of vision, covered the injured eye with a dirty piece of cloth, which he picked up from the ground. Eleven days later upon arising from bed he felt his mouth closed, more on the left side, and as the hours passed, pains which passed to the left arm, and disturbance of deglutition. By afternoon he was unable to swallow solid food, only liquids. He spent the night restlessly and suffered continuous nightmares. This condition remained unchanged for about three days, when he felt suffocation at night and difficulty in breathing. On the 27th of August he called at the clinic.

Examination of face. The left half of the face was immobile, without expression, and without frontal wrinkles, which, however, were visible on the right. The supercilium was lowered. The oral fissure was less closed on the right side, being displaced toward this side; the left nasolabial line could no longer be noticed. The patient was unable to whistle, due to the absence of the labial movements on the injured side. In this, as in the previous case, there was a facial paralysis on the side of the injury, left blepharoptosis and left unilateral trismus.

Ocular examination. As in the former observation, there was atrophy of the ocular globe as a consequence of the rupture of the cornea and of the sclerotic, with loss of vitreous. There was sensitivity of the ciliary body upon pressure. The cornea presented a rupture in its upper third and frank inflammatory phenomena. The lacerated sclerotic was in the process of cicatrization. The bulbus was in frank atrophic progress. We saw nothing on the eyelid except a slight erosion on the external surface. There was a blepharoptosis on the side of the ocular injury, which denoted a paralysis of the 3d pair in addition to that of the 7th, the paralysis being on the same side as the injury.

Additional examinations: Gait normal; sensibility, normal; Romberg, negative; Ruine and Weber tests, negative; reflexes—present on the right, absent on the left; Masseter's abolished, due to trismus; plantars and abdominals present, as well as biceps, triceps, patellars, and Achilles. Babinsky and variants, absent. Temperature—no change in the thermal curve.

Evolution and treatment. Until August 31st there was no change in the progress of the tetanus, which continued with all the original symptoms, although local treatment of the focus had been instituted with various antiseptics, such as eye washes, mercury oxycyanide 1:5,000, and applications of bioxide of hydrargyrius, 2 percent.

On September 1st, the application of antitetanic serum was begun in doses of 5,000 units, subcutaneously, and the parenteral injections in addition to the local treatment. This therapy was continued for four more days without any change in the patient's condition. This rather tended to grow worse, since he could no longer take food of any kind, even liquids, which caused vomiting and choking due to dysphagia. No other symptoms appeared in the direction of the trunk or limbs, the tetanus remaining localized in the cephalic region.

On September 3d, injections of 3-percent phenic acid were started, 3 c.c. subcutaneously twice a day, because of the excellent results obtained in case 1. The parental injections of 5-percent sodium bicarbonate were continued, also long warm baths, the patient being kept quiet and in semidarkness. On the 10th there was scarcely any noticeable change in the condition; the patient was still unable to take food. Intravenous injections of electrargol were then instituted, since excellent results had been previously obtained, the dosage being 10 c.c. twice a day, which relieved the patient greatly in regard to the contractions of the muscles of the face and even improved his general condition. These were therefore continued, accompanied by injections of phenic acid or magnesium sulphate, 10 c.c. daily of a 20-percent solution. From the 20th on the progress of the disease took a different turn, for the facial paralysis and the blepharoptosis began to recede; however, the trismus remained, although much reduced; the patient was now able to open his mouth. The dysphagia disappeared as well as the attacks of suffocation. On October 10th the patient was free from the paralysis of the facial and of the oculomotor, but he still presented a slight trismus, which now was more visible on the left side.

The patient was discharged on the 20th, still presenting a slight trismus and atrophy of the ocular globe. Three months later I again saw the patient, who was then entirely free from the trismus and hence cured.

Case 2 presents an interesting factor not found in the first observation; namely,

the blepharoptosis on the same side as the ocular injury. This fact had already been reported by Hadlich and Solmsen.

Hernani Alves, in his thesis in 1910, cited blepharoptosis in one case on the opposite side to the injury and in another case on the same side.

References

Jayme-Goyaz, G. G. Thesis, Rio de Janeiro, 1925; Gaz. d'Hop., Rio de Janeiro, 1888; Rev. Cirur., Rio de Janeiro, 1888.

Albert. Thèse de Lyon, 1890.

Jannin. Thèse de Paris, 1892.

Houques. Thèse de Bordeaux, 1894.

Le Dard. Thèse de Paris, 1896.

Wellner. Rev. de Neurol., 1898.

No Haltenhoff. Anal. de la Suisse Romande, 1902.

Worms. Thèse de Lyon, 1904.

Poan de Sapincourt. Thèse de Paris, 1904.

Achard, C. Clin. med. de l'Hop. Beaujon, 1923.

Guimarães, J. P. Rev. dos cursos prat., Rio de Janeiro, 1893.

Alves, H. F. Thesis, Rio de Janeiro, 1910.

Chappé, A. Traité d'Opht., 1939, v. 8, p. 409.

Wiolin. Zur Behandlung des Tetanus. Wratschebnaja Gaseta, 1914, v. 21, no. 8. In Zentralbl. f. d. ges. Ophth., 1914, v. 1, p. 224.

Castellain and Lafargue. Du tetanos consécutif aux lésions oculaires. Ann. d'Ocul., 1920, v. 157, no. 1, p. 9.

Van der Hoeve. Tetanus bei Augenverwundung. Neder. Tijd. v. Genees., 1920, v. 64, p. 1173.

Addario la Ferla. Due casi di infezione tetanica per traumatismo della cornea di cui uno sequito da esito mortale. Lett. Oftal., 1931, v. 8, p. 10.

Bonnet, Paufique, and Perron. Tetanos ophtalmoplégique guéri. Bul. Soc. Opht., Paris, 1933, no. 9.

Gauthier, M., and Rouvier, J. Un cas de tetanos aigu traité par la méthode de Dufour et suivi de paralisie post-serothérapique partielle de la III paire. Bul. Soc. méd. Hop. Paris III, 1937, v. 53, p. 386.

Jebavy. Ein Fall von Tetanus cephalicus. Ocni Klin. Uuiv. Brno. In Zentralbl. f. d. ges. Ophth., 1937, v. 39, p. 124.

Groenouw. Beziehungen der Allgemeinleiden zu Veränderungen des Seeorganes. Graefe-Saemisch Handbuch der gesamtem Augenheilkunde. Leipzig, 1904, v. 11, p. 616.

Schneider, E. A. Le tetanos consécutif aux lésiones oculaires. Ann. d'Ocul., 1916, Sept., p. 395 (19 cases of which only 3 were cured).

Discussion

Dr. V. G. Casten (Boston, Massachusetts): I wish to congratulate Dr. Jayme for his excellent results in the two cases reported before this Congress.

General symptoms of tetanus following perforating wounds of the eyeball begin on the sixth to eighth day, and at first show the cephalic type of tetanus with lockjaw, facial paralysis, and ophthalmoplegia. The convulsive state then follows. It is a severe type of infection, death occurring in over 75 percent of the cases the end result of which has been stated. Less than 30 cases of cephalic tetanus are recorded in the world's literature; half of these were reported before tetanus antitoxin came into general use.

The type of injury and causal agent are very significant, according to the report by Cogan in the American Journal of Ophthalmology (1939); at least 35 percent were due to horsewhip injuries and most of the others to foreign bodies associated with the barnyard. Panophthalmitis

of a very severe endophthalmitis is usually present. It would seem that tetanus was more likely to develop in patients showing mixed infections.

By the time panophthalmitis has appeared the tetanus infection has extended beyond the eye, and consequently enucleation is of little value.

In one case enucleation was done on the day of injury but death followed from tetanus. In eight of the cases reported the eye was enucleated but only one patient recovered. Antitoxin was not used in any of this series.

At the present time tetanus toxoid, two weekly intramuscular injections, provides a lifelong immunity. Pediatricians are beginning to give it combined with diphtheria antitoxin to children. In the present war this has been found to be very successful in the case of both man and horses.

THE ROLE OF HOME TRAINING IN ORTHOPTICS*

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It is the purpose of orthoptic training to establish or reëstablish normal binocular functions. It is the problem of the ophthalmologist and the orthoptic technician to achieve this end as efficiently as possible. The problem, however, is complicated by the fact that it is more than a pair of eyes with which they are dealing. Psychological factors, transportation difficulties, financial limitations, and such cannot be disregarded. Therefore, daily training periods conducted by a qualified orthoptic technician in the office or orthoptic clinic are the ideal that can be realized in only a few isolated cases. As a compromise measure, less frequent supervised-training periods may be supplemented by home exercises in suitable cases, for those capable of carrying them out.

Home training under these circumstances can assume an important role in an orthoptic program. If carefully planned and conscientiously carried out, it can contribute greatly to a satisfactory end result. If prescribed in haphazard fashion and executed in similar manner, another failure may be ascribed to so-called orthoptics, a misconception and a misnomer under such circumstances. Cases in which the patients can be trained at home and the type of home training suitable for them demand consideration.

These cases divide themselves almost automatically into the phorias and the tropias in accordance with the two major groups of ocular-muscle imbalances. The phorias already endowed with binocular vision present the simpler problem. Furthermore, in those cases with no history of squint, symptoms indicating the necessity of orthoptic treatment seldom develop until maturity has been reached. The tropias, on the other hand, are manifest in early childhood as a rule. With few exceptions, sufferers from squint must be taught first what binocular vision is. The squint must be converted into a phoria, possibly with the aid of surgery, and sufficient reserve power must be developed so that there will be no reversion to the squinting condition.

Since the greater number of cases with

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which the orthoptic technician deals are squints at the inception of treatment, I want especially to stress what can be done in the way of home training for the tropias.

In most cases of monocular squint the patients suffer from amblyopia ex anopsia if their squint has existed for any length of time. The accepted treatment of this condition is occlusion of the better eye. This is a passive form of home training. If the visual acuity of the emblyopic eye is low, 20/70 (6/20) or less, occlusion must be complete and constant, of such a type as to prevent any peeking. If the vision is 20/50 (6/15) or better, sufficient for the ordinary tasks of daily life, the incentive to peek is not so compelling, and less drastic occlusion such as opaque paper or translucent music mending tissue pasted on the lens can be adopted. After normal vision has been restored, part-time occlusion, a given number of hours a day or week, can be continued in order to prevent a relapse of amblyopia. This is advocated by Peter[1] and by Travers.[2]

Alternating squinters should alternate the occlusion from eye to eye or, if convergent, they may wear the inner half of each lens occluded (half occlusion).

Finally, there is a group of patients in whom binocular vision has been restored, but who manifest macular suppression of the poorer eye. For these individuals, some form of fogging of the lens of the better eye is advisable; such as, a Chavasse glass, a clip-on smoked glass (Miss Pugh has worked out a series of these of varying densities), cellulose Scotch tape, colorless nail polish stippled on the glass, or a red filter.

The necessity for providing exercises or tasks to be done with the amblyopic eye has been stressed by Hardy[3] and by Travers.[2] According to the former, "It is a great mistake and an invitation to failure to assume that because the better eye is covered the child is striving to improve the vision of his amblyopic eye. Such perfunctory treatment is the probable explanation of much dissatisfaction with results. An infant can hardly be expected to comprehend the reasons for depriving him of the use of his better eye. He may lack the knowledge that better vision than he has is possible, certainly the knowledge as to how to go about improving it. In a world controlled by adults much must be accepted on faith and the child is apt passively to accept what he does not understand.

"For these reasons, it is important to encourage and stimulate him in every possible way to use the eye for clearer and more critical observation."

Travers has stated, "It is desirable to exercise the amblyopic eye as much as possible in order to improve its vision rapidly. It is not sufficient simply to tie up the better eye and leave the child to his own devices. The parents should provide him with tasks which will call for a certain amount of discrimination."

According to my experience, it is not sufficient merely to instruct the parents to have the child color, trace, string beads, et cetera. More satisfactory results are obtained if the ophthalmologist or his technician plans the amblyopic exercises and furnishes the material to be worked on between office visits. To be required to bring back and show a piece of work well done provides an incentive for careful execution. Furthermore, the material can be adapted both to the visual capacity and the stage of mental development of the child. As both advance, tasks can be increased proportionately in difficulty.

For this purpose, I keep on hand a supply of coloring, tracing, cut out and pasting, dot drawing books and such, as well as beads for stringing, sewing cards, games, and puzzles of various sorts. Noon

hours are frequently spent searching toy and children's book departments for new material and the ten-cent store is an unfailing source of supply.

School work is an automatic source of stimulation for the amblyopic eye; hence it may be argued that special work need not be required of the school child. Even though this is true, it is occasionally advisable, as for example when the occlusion has been reduced to part time, to supplement school work with planned amblyopic exercises.

The primary requisite for binocular home training is normal retinal correspondence. Present-day authorities on orthoptics have so stressed the importance of diagnosing abnormal retinal correspondence and the attendant dangers of unsupervised orthoptics that the statement —Any binocular home training is contraindicated in the presence of abnormal retinal correspondence—is now almost axiomatic.

In addition to normal retinal correspondence, a patient suitable for home training should demonstrate some ability to fuse images at his angle of deviation on an instrument providing satisfactory objective control, and he should have some range of fusion.

In an anlysis of the exercises and instruments suitable for home training, the stereoscope of the Holmes type demands first consideration, since it has long been the instrument of choice for this purpose. It is relatively simple, compact, and inexpensive, and a variety of charts have been designed for its use. Its limitations, on the other hand, along with those of the patient using it have not always been appreciated.

With its decentered lenses and a possible variation in the separation of the targets (identical elements) from 25 mm. to 135 mm., a prismatic effect ranging from approximately 28$^{\Delta}$, base out, to 28$^{\Delta}$, base in, can be obtained at the principal focus of the lens. (These calculations are based on an 80-mm. separation between lens centers and a 60-mm. interpupillary distance.)

Many of the charts designed for ocular exercises, such as the Kroll, Cruise, and Wells charts, with the exceptions of the H and I series, have a separation of 60 mm. requiring about 10$^{\Delta}$ of convergence for fusion. To prescribe such a set of charts, as has been done, for a person with convergent squint of 30$^{\Delta}$ with poor fusion and little amplitude of fusion, who must diverge relatively 20$^{\Delta}$ in order to combine the pictures, is unwarranted optimism. To do so for a person with divergent squint of 30$^{\Delta}$ who has little converging power is likewise "an invitation to failure," if I may borrow Doctor Hardy's phrase.

It is essential that the charts prescribed be within the fusion range of the patient. What this range is in millimetres can be determined satisfactorily with split charts used on one of the modifications of the stereoscope provided with a holder for their use. Accessory prisms may be added to the stereoscope if deemed advisable for obtaining fusion. They add to the expense and increase distortion, and the problem of a satisfactory clip for holding them, especially the stronger ones, has not been solved to my knowledge.

Two methods may be used in exercising with the stereoscope. Charts of increasingly difficult separation (wider in convergent, narrower in divergent cases) may be attempted, or the slide may be moved toward or away from the eyes, thus introducing variations in the accommodation relationship. Convergent-strabismus patients find it difficult to maintain fusion when accommodation is brought into play by bringing the chart toward the eyes, and they should strive to hold fusion by so doing. Conversely, those with divergent strabismus find fusion facilitated

with the introduction of accommodation, and exercising in the reverse direction is indicated for them.

The subject matter of the charts must also be considered. It must be within the comprehension of the patient and should appeal to his interest. The Wells ONE series for example, excellent for increasing diverging or converging power according to the direction used, could not be expected to elicit much attention from a four-year-old.

A pitfall of which to beware in the case of young children is that of memorizing the charts and learning the right answers. The check marks of the Keystone view charts are identical throughout a given series, and the lines may be reported as crossed, whether or no. Too frequent exposure to a given chart may result in correct reporting without attentive looking. In order to guard against or minimize the possibilities of this memorizing on the part of the young patient, I keep a supply of various charts on hand and loan him the requisite number (a half dozen or so) to be worked on between office visits. In fact, I run a sort of chart circulating library which I have augmented by charts of my own making.

Besides the advantage of providing variety, this system permits increasing the difficulty of the material as the eyes improve. I believe that it would work satisfactorily in any orthoptic clinic or office where a large amount of orthoptics is done, and I commend it to your consideration.

In prescribing home exercises, the various adaptations of the stereoscope should not be overlooked, such as the correct-eye-scope, designed for use with split charts, the kinetic stereoscope with its provision for moving targets, and the so-called *novelview* and *tru-vue* types designed for use with films. As with the Holmes stereoscope, the limitations and advantages of each should be analyzed in relation to the ability of the patient before homework is undertaken.

The Remy separator which may be briefly described as a lensless stereoscope deserves more than mere mention. It affords an excellent home exercise for patients with either divergent or convergent imbalance, who have some ability to bring their eyes toward parallelism and who are capable of making the effort to do so, in general an age group beginning at 10 years.

It has the advantage of being so simple that it can be made at home, and the small boy who constructs one will be anxious to demonstrate to his own satisfaction that it really will work. For the clinic patient forced to count pennies, this instrument has an added advantage in the minimum of expense involved in obtaining one.

In England, the cheiroscope is an even more popular instrument for home training than the stereoscope. The compact English model is rented by opticians at the rate of 10 shillings ($2.50) for a period of three months. Accessory prisms are often introduced to compensate for high deviations. With proper coöperation from the opticians, a similar system ought to work out advantageously in this country. Mosaic patterns and blocks used with the cheiroscope can be helpful in stimulating fusion, and tracing exercises in breaking down suppression and amblyopia and making the patient aware of his two eyes.

One of the simplest exercises that can be prescribed for a convergent squint is that known as "recession with a red glass." Since the visual axes of a convergent squint cross within optical infinity, the images of an object placed at their intersection should fall on the two maculae and be fused. With a red glass or filter held before one eye, and a small light placed at the crossing of the visual axes, the red light seen by the one eye and the white light seen by the other should fuse

to make a pinkish light. (Appreciation of a deep red light or a white light only, indicates suppression of one eye.) Once the images are combined at this point, it may be possible to maintain fusion for a short range when the light is slowly receded from the eyes, and to increase this range with repeated efforts.

This recession exercise is also excellent for patients with divergent squint based on a divergence excess, who have good converging power for near, but who manifest a deviation on looking in the distance.

Not only is this a simple exercise, but one in which a daily record of achievement can be kept, thus affording a check on progress and execution valuable to the ophthalmologist or technician and stimulating to the patient. In order to prevent lapses of attention and perfunctory performance on the part of the patient, the importance of checking on this progress and verifying the record must not be overlooked by those supervising the orthoptics. (I will be glad to supply a set of directions and record sheet which I have found practical and would like to add that red kodaloid obtainable from the Eastman Kodak Company provides an inexpensive and satisfactory red filter. This comes in sheets one foot square and can be cut to the size desired.)

When fusion can be maintained with the red glass to 6 meters (20 feet) and held with ease at this distance, the phoria stage should have been reached and the patient be ready for prism exercises, if they are deemed advisable—base in for convergent tendencies and base out for divergent. Dr. Conrad Berens has long been a proponent of prism exercises for the phorias and I refer you to his article (in collaboration with Drs. Losey and Hardy[4]) for detailed descriptions and directions.

Bar reading, which may be done with a pencil held a few inches in front of the printed page, or with a specially devised bar, is an excellent home exercise to be recommended for certain of the phorias; for a patient with convergence excess whose eyes tend to overconvergence when accommodating, or for one with convergence insufficiency whose eyes tend to divergence when looking at a near point. It is particularly to be recommended for a condition of slight imbalance accompanied with a persistent macular suppression of one eye.

No article on orthoptic home training would be complete without mention of convergence on a pin, line, light, finger, or pencil as the classical exercise for convergence insufficiency. The technique for this is so familiar that I believe that it requires no elucidation. Psychological factors, however, are particularly important. This excellent exercise is so simple that the patient is apt to minimize its importance and to be skeptical regarding its efficacy. He must be impressed by strong admonition and the full weight of authority of the necessity for carrying out "something silly" according to his interpretation.

It is not within the scope of this paper to enumerate all the possibilities of home training in orthoptics, or to point out all the attendant advantages, limitations and pitfalls. Once attention has been called to the major ones, the alert ophthalmologist or technician interested in the problem will readily detect minor ones. If he or she is endowed with "imaginuity" (again I borrow a phrase from Doctor Hardy) present techniques may be improved, new methods developed, new material adapted to make orthoptics more efficient in achieving its desired end, normal binocular vision.

At the risk of being too didactic, may I summarize in conclusion the following general rules to be observed whenever orthoptic home training is to be recommended:

1. All patients attempting binocular home training must have normal retinal correspondence.

2. Every binocular exercise prescribed should provide an individual check for each eye, in order to insure simultaneous functioning.

3. The task prescribed should be within the ocular capacity of the patient.

4. The task prescribed should likewise be within the mental capacity of the patient.

5. Explicit directions for carrying out the homework are essential. Any written directions must be supplemented with oral ones. This holds true even for intelligent adults with phorias.

6. Checks must be made on subsequent office visits to determine that directions have been understood and are being properly carried out.

7. As progress is manifest, the difficulty of the homework outlined should be increased proportionately.

In my experience, when these rules are observed and home training conscientiously carried out in conjunction with office or clinic orthoptics, the results are sufficiently gratifying to compensate for the additional time and thought required in supervising this form of orthoptic treatment.

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References

[1] Peter, L. C. Trans. Ophth. Soc. U. Kingdom, 1932, v. 52, p. 325.
[2] Travers, T. aB. An essay on concomitant strabismus. Monograph published by Brit. Jour. Ophth., 1936.
[3] Hardy, L. H. Paper read before the College of Physicians of Philadelphia, Section on Ophthalmology, March, 1933.
[4] Berens, C., Losey, R. L., and Hardy, L. H. Routine examination of the ocular muscles and nonoperative treatment. Amer. Jour. Ophth., 1927, v. 10, no. 12, pp. 910-918.

Discussion

Dr. Conrad Berens (New York). I agree with Miss Stark that home training is exceedingly important provided the parents, children, or patients are sufficiently intelligent and can be made to realize the importance of what they are doing and have the tenacity of purpose to work hard as well as intelligently.

Miss Stark has had such long and varied experience in this entire field that I am sure she will agree with me, that the "phorias" are not only a "simpler problem" but offer the much greater field for benefit. I would like to ask Miss Stark whether even patients, who have so-called orthophoria and who fatigue rapidly or have other asthenopic symptoms, are not often greatly benefited in certain instances by home training.

Miss Stark speaks of orthoptic training in heterotropia as a possible aid to surgery. It is increasingly our belief, especially after a recent study of 324 surgical cases of heterotropia treated at the New York Eye and Ear Infirmary, that in most cases you cannot expect too much correction of the deviation by orthoptic training alone but that pre- and postoperative orthoptic training improves functional results.

It has always been my contention that we possibly expect too much from orthoptic training. It is also my experience that unless good binocular vision and fusional amplitude can be obtained, the eyes even though in fairly good position, quickly lapse into bad habits and undo much of the work that can be accomplished by orthoptic training.

I do not believe that the point made by Miss Stark concerning the need for using the amblyopic eye on prescribed exercises

can be too strongly insisted upon; to my knowledge, 20 years ago, Duane prescribed cards that had to be threaded with wool and other forms of close work requiring critical seeing by the amblyopic eye. Although Miss Stark speaks of not being able to use the eyes unless the visual acuity is a certain amount, with clip-on lenses and even telescopic lenses we can make the use of the amblyopic eye more interesting for the average person and even for some young children.

My personal feeling is that reading the finest print that the patient is able to see, is one of the best and most interesting exercises that can be given to stimulate the amblyopic eye. For those who believe that flashing and intense illumination are important, a flasher may be attached to an electric light socket and a strong bulb placed close to the reading material.

I am sorry that Miss Stark has not gone into detail concerning home training in heterophoria and orthophoria, because those technicians who work in physician's offices should have a great deal of this type of training to supervise. Moreover, now that ophthalmologists are becoming more interested in squint training, as evidenced by 10,378 visits during 1939 to the Orthoptic Clinic at the New York Eye and Ear Infirmary, an increasing amount of work is being done on patients who have heterophoria. Obviously it is unnecessary and too expensive to send patients who are willing to coöperate to a clinic or to the office more often than necessary for treatment of heterophoria, but you should be certain that the patient is carrying out instructions properly before he is left to his own devices.

In heterotropia, it is obvious that, whenever possible, so-called normal retinal correspondence should be obtained before instituting home treatment.

We cannot insist too strongly on interesting cards adapted to the age of the patient being treated. No matter how interesting the cards are at first, they must be changed in order to sustain interest. Fortunately, there are many different sets of inexpensive stereoscopic cards now available at not too great a cost to the patient.

Clinic patients often cannot afford to buy stereoscopes, but some training can be done by cupping the hands over the nose, especially in divergent squints with normal retinal correspondence, and placing the cards on the table. If one eye is amblyopic the target can be larger on one side than on the other and the light may be increased.

I am glad that Miss Stark has spoken of exercises by approximation and recession using a red glass at first; although she does not mention prisms and their use in this exercise, I know that she is familiar with this type of training. If the patients have normal correspondence, it is a useful exercise, but it does not stimulate extramacular fusion. Therefore, as soon as possible the jump prism exercises should be combined with this exercise. My personal feeling is that it is usually a mistake to discharge the patient until he has good amplitude of convergence, divergence, and supravergence with prisms, using no red glass or anything else to dissociate the images of the two eyes. Patients should also be able easily to maintain fusion at the near point while reading fine print, if possible 300-mm. print. An important feature of the recession exercise is that it is easy to explain to the patient with convergent squint and to the family that when he can walk across the room and keep the images together, the squint is cured. However, as soon as you find you are not making progress, everything else being equal (and naturally refractive errors having been carefully corrected), it is time to consider seriously placing the eyes within their fusion range by operation.

I have had little success with bar read-

ing, but for the limited use which Miss Stark suggests it is practical.

Miss Stark speaks of converging exercises on a pin and on a line if hyperphoria is present. This is an excellent exercise. By permitting the patient to relax and fuse as well as by following the pin in toward the nose or away from the nose in the case of convergent squint or convergence excess with marked esophoria, you can stimulate extramacular fusion, approximating what can be done with the jump method of prism exercises.

Those who have had little experience with home training in heterophoria and heterotropia should place Miss Stark's conclusions in a conspicuous place; I am sure that the more experience they have with this form of treatment, the more they will appreciate the importance of frequent supervision of this type of exercise and, if possible, have the work in the home carried out under the supervision of trained technicians. We have had success in two patients whose parents purchased a major amblyoscope for home use.

Miss Stark did not mention having trained technicians go with the patients to their homes immediately after operation. In some of our most outstanding successes, even before we appreciated the importance of abnormal retinal correspondence, I believe our results were greatly influenced by constant home supervision for a period of two or three months. At that time we had practically no trained technicians and had to rely upon nurses who were but superficially trained in this work.

I would like to add one point to Miss Stark's conclusions and that is the psychological importance of inspiring the patient with your belief in what you are doing and if possible let the patient see what has been done for similar cases. It is only by constant supervision and by repeated stimulation that the necessary coöperation can be obtained.

I have benefited greatly by Miss Stark's scholarly exposition of this important adjunct to the therapy of motor anomalies. Moreover, I am sure that because of her paper the subject of orthoptics is clarified and our patients will receive better attention in the future.

NOTES, CASES, INSTRUMENTS

MONILIAL CONJUNCTIVITIS

RUSSELL F. MADDREN, M.D.
Hackensack, New Jersey

Monilial conjunctivitis has been noted so infrequently that reporting a single case appears justifiable. Fazakas,[1] the Debrecen ophthalmologist, cultured material from 1,791 eyes and grew fungi 608 times. In only four instances were the organisms found identified as Saccharomyces or as Monilia. He does not state from what part of the ocular apparatus he cultured Monilia. Forty-one years ago Pichler published two remarkable cases, and in 1927 Norton[2] reported another from Seoul, Chosen. In five years' study of conjunctivitis of the newborn, Thygeson[3] has never observed monilial conjunctivitis despite high incidence of thrush in the cases he investigated by smear and culture. Duke-Elder[4] states that fungus infections of the conjunctiva are rare and that diagnosis rests entirely on the identification of the organism concerned.

Monilia albicans (Zopf: Die Pilze, 1890) is identical with Oidium albicans described by Robin (Hist. Nat. Vég. Paras, 1853). It is not encountered on normal skin[5] and by culture and subculture on appropriate media it can be conveniently and certainly differentiated from other levuriform organisms which apparently are unimportant saprophytes.

Case report. A 59-year-old woman presented herself at the office on December 14, 1940, complaining of an extensive rash beneath the breasts, in the groins, in the concavities of the pinnae, and in the external ear canals. She related that the eruption had started beneath the breasts eight years previously, thence had progressed to the groins, and recently had involved the ears. Inspection revealed slight perlèche and a few characteristic flat pustules scattered adjacent to the intertriginous eruption. During the examination, moderately severe angular conjunctivitis was noted. Material was scraped from the tongue and from three different places on the skin and was planted on Sabouraud's medium. The clinical diagnosis was moniliasis, and treatment was instituted by exposing each ear to 50r of unfiltered radiation, the prescribing of a mild fungicidal evaporating lotion, and the ordering of vitamin-B complex, to be taken in moderately large doses three times daily.

When the patient returned on December 21st, Monilia were growing in all of the cultures. Further study revealed these organisms to be M. albicans. The patient's skin lesions had improved, but her conjunctivitis was no better. Upon questioning, she related that it had been present for many weeks. She had gone to an optometrist, who had fitted her with glasses some years previously. He had changed her lenses, but the change had afforded no relief.

In view of this woman's extensive and long-standing cutaneous moniliasis, possible involvement of the conjunctivae was suspected, notwithstanding the absence of marginal blepharitis and the absence of any skin lesions either upon or adjacent to the eyelids. The left lower lid was partially everted by drawing down the skin just below it and then a heat-sterilized platinum loop was cautiously passed to the bottom of the conjunctival sac, moved about a little, and withdrawn before the traction on the lid was released. The scanty material obtained was smeared upon a slant of Sabouraud's medium and put to incubate at room temperature. In similar manner a culture was made from the right eye.

Within one week three colonies of Monilia were growing on one slant and five colonies on the other.

On December 28th, and on January 4, 1941, cultures were made from each conjunctival sac. They also became positive for M. albicans within eight days. Diagnosis was confirmed by Mary E. Hopper, M.S., New York mycologist.

The patient was well satisfied with the improvement in her skin condition and flatly rejected repeated advice to consult an ophthalmologist and so, on January 4th, after the third set of cultures had been made, the following solution was prescribed with directions to instill two drops into each eye three times a day:

℞

Sod. benzoat.	2%
Sod. salicyl.	1%
Sod. chlorid.	0.82%
Aquae rosae	
Aq. camphorae ana q.s.	

On January 18th, the conjunctivae were clear, and cultures made from the eyes in the same manner as before failed to show any growth of Monilia in 14 days. One is reluctant to believe that two weeks' use of a faintly alkaline (pH 8.3) and mildly fungicidal collyrium designed to be isotonic with tears could have been effective by itself. It should not be overlooked that relief of conjunctival symptoms occurred in a patient whose cutaneous moniliasis was yielding to vigorous systemic and local treatment.

On January 25th, the patient was discharged cured. All the skin lesions had resolved, and there was only slight residual pigmentation where intertrigo previously had been present.

Comment. Moniliasis is seen not infrequently by dermatologists. It might be that closer collaboration between physicians who treat skin diseases and those who treat diseases of the eye would result in demonstrating that monilial conjunctivitis is less rare than has been supposed.

372 Union Street.

References

[1] Fazakas, A. Ueber die Bedeutung der Pilze für Augenheilkunde. Zeit. f. Augenh., 1938, v. 94, March, p. 253.

[2] Norton, A. H. Thrush of the conjunctiva. Amer. Jour. Ophth., 1927, v. 10, May, p. 357.

[3] Thygeson, Phillips. Personal communication.

[4] Duke-Elder, W. S. Textbook of Ophthalmology. Saint Louis, C. V. Mosby, 1938, p. 1654.

[5] Benham, R. W., and Hopkins, A. M. Yeastlike fungi found on the skin and in the intestines of normal subjects. Arch. Dermat. and Syph., 1933, v. 28, Oct., p. 532.

Lewis, G. M., and Hopper, M. E. Infections of the skin due to M. albicans. N. Y. State Jour. Med., 1938, v. 38, June, p. 11.

ON OPHTHALMIC LENS TRANSPOSITION*

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New York

In current books on refraction and practical optics one still finds many rules given for the ordinary transposition of lenses. It seems unnecessary to clutter one's mind with many rules when the matter can be made much simpler. There are only four transpositions (aside from oblique cross cylinders), and three of these can be done with one simple rule, the fourth with another simple rule. The three transpositions covered by one rule are: (1) sphero cylinder to sphero cylinder; (2) simple cylinder to sphero cylinder; (3) cross cylinder to sphero cylinder.

The rule in skeleton form is a five-word

* From the Department of Ophthalmology, Stuyvesant Polyclinic Postgraduate School.

(five-finger) rule "Sum (for) Sphere, Change Sign (and) Axis." In more elaborate form, which need never be remembered once the skeleton rule is understood, the rule is: Given a sphero-cylinder, "take the sum of the sphere and cylinder for the new sphere, keep the cylinder value but change the sign of the cylinder from plus to minus or from minus to plus, change the axis of the given cylinder by adding 90 degrees or subtracting 90 degrees." Sum of course means algebraic sum in the same sense as adding plus and minus spheres.

Applying the five-word rule to (1) Sphero cylinder to sphero cylinder; for example,

+1.50 D. sph. ⊂⊃ —.50 D. cyl. ax. 90°,
gives
+1.00 D. sph. ⊂⊃ +.50 D. cyl. ax. 180°.

Applying the same five-word rule to (2) Simple cylinder to sphero cylinder: One turns the simple cylinder into a sphero cylinder by supplying mentally (or on paper at first) a zero sphere. Thus +1.00 D. cyl. ax. 90° becomes 0.0 D. sph. ⊂⊃ +1.00 D. cyl. ax. 90°. Using the same five word rule (the sum of 0.0 and +1.00) for sphere, and so forth, gives +1.00 D. sph. ⊂⊃ —1.00 D. cyl. ax. 180°. Similarly, —1.50 D. cyl. ax. 70°, becomes 0.0 D. sph. ⊂⊃ —1.50 D. cyl. ax. 70°; which gives by the same rule —1.50 D. sph. ⊂⊃ +1.50 D. cyl. ax. 160°.

Applying the same rule to (3) Cross cylinder to sphero-cylinder: We simply transpose the first cylinder by the five-word rule and add the second cylinder. For example, given, +1.50 D. cyl. ax. 90° ⊂⊃ —1.00 D. cyl. ax. 180°, we transpose the first cylinder by supplying a zero-sphere thus 0.0. D. sph. ⊂⊃ +1.50 D. cyl. ax. 90°; which gives +1.50 D. sph. ⊂⊃ —1.50 D. cyl. ax. 180°, and add —1.00 D. cyl. ax. 180°. Result: +1.50 D. sph. ⊂⊃ —2.50 D. cyl. ax. 180°.

Thus by noting a few small points easily remembered when understood, one can do the three most common transpositions by the application of the simple five-word rule.

The fourth transposition, (4) Sphero-cylinder to cross cylinder, is best done by remembering that every sphere may be regarded as two equal-powered cylinders at right angles; for example, +1.00 D. sph. equals +1.00 D. cyl. ax. 90° ⊂⊃ +1.00 D. cyl. ax. 180°, or +1.00 D. cyl. ax. 80° ⊂⊃ +1.00 D. cyl. ax. 170°, and so on.

The rule for sphero-cylinder to cross cylinder then becomes: Break up the sphere in two cylinders and add the given cylinder; for example, +1.00 D. sph. ⊂⊃ +.50 D. cyl. ax. 90°, gives + 1.00 D. cyl. ax. 90° ⊂⊃ +1.00 D. cyl. ax. 180°. Add +.50 D. cyl. ax. 90°. Result: +1.50 D. cyl. ax. 90° ⊂⊃ +1.00 D. cyl. ax. 180°. Or —2.00 D. sph. ⊂⊃ +1.50 D. cyl. ax. 70° gives —2.00 D. cyl. ax. 70° ⊂⊃ —2.00 D. cyl. ax. 160°; Add +1.50 D. cyl. ax. 70°. Result —50 D. cyl. ax. 70° ⊂⊃ —2.00 D. cyl. ax. 160°.

In practical teaching I have found these methods, one five-word rule for the three transpositions and one other short rule for the fourth transposition, the easiest to remember and to apply. They work equally well for all combinations of plus on plus, minus on minus, plus on minus or minus on plus.

37 West Ninety-seventh Street.

THE MOST USEFUL AND INEXPENSIVE OPHTHALMIC KNIFE

CHARLES A. BAHN, M.D.
New Orleans, Louisiana

Too often rusty and dull knives are used in ophthalmic offices and clinics. Styes, chalazia, pterygia, conjunctival and corneal foreign bodies, and so forth, are frequently operated upon with dull and rusty knives that belong in the trash can

—not in human eyes. Certainly, ophthalmic surgeons would not want their own eyes touched with such instruments.

Unfortunately, it has been impossible to purchase a knife that could and should prevent this misuse of worn-out cataract knives, and the like. My experiments to remedy this evil in a simple, practical way have brought out these fundamentals: 1. The handle must correspond in size, shape, weight, and balance with other knives used by ophthalmic surgeons to facilitate maximum dexterity. 2. Blades of different types should be adapted to a universal handle. For foreign bodies, a sharp-pointed blade with a gradually increasing belly is of advantage. 3. Both blades and handle must be easily obtainable. 4. The handle must cost much less than cataract or similar ophthalmic knives and blades, but a few cents each.

I constructed a knife based on these requirements. It has been used hundreds of times in my office and has proved of great practical value. The manufacturers, the Bard-Parker Company of Danbury, Connecticut, to whom this model was submitted, had made a small quantity of experimental handles. This is their Special Eye Handle, no. 9, and costs $1.50. The no. 11 blade, for the removal of foreign bodies, is longer and triangular. The small scalpel blade is best adapted for pterygia, chalazia, and other minor operations. Both cost but a few cents each.

Fig. 1 (Bahn). Simple, practical, ophthalmic knife.

In view of the rapidly rising costs of ophthalmic knives and other equipment, and increasing difficulties in obtaining them, ophthalmologists will make no mistake in procuring this Bard-Parker Special Eye Handle no. 9 with the blade they prefer. It is the most practical, useful, and inexpensive knife yet devised.

1703 Pere Marquette Building.

SOCIETY PROCEEDINGS

Edited by Dr. Ralph H. Miller

CHICAGO OPHTHALMOLOGICAL SOCIETY

May 13, 1940

Dr. Earle B. Fowler, *president*

Preretinal hemorrhage

Dr. Roland I. Pritikin said that C. G., a negress aged 57 years, stated that on awakening several weeks ago she had a sensation of "skin" forming over the left eye; a clouding sensation, as if something were growing to the outside. There was no associated pain, tearing, nor headache. Visual acuity of the left eye since then has been progressively worse.

Vision R.E. was 20/20; vision L.E. was reduced to the ability to count fingers at 8 inches.

The left eye showed a large deep-dark mass, 4 D.D. in diameter, in front of the macula, which was interpreted as a large preretinal hemorrhage due to underlying low-grade chorioretinitis and a sclerosed vessel.

Transillumination was difficult and not informative.

Left inflammatory central macular hole

Dr. R. I. Pritikin presented E. W., a medical student aged 25 years, who had noticed a blurring and distortion of the central vision in his right eye in December, 1939. A recent refraction had shown 20/12+ vision in each eye with +0.75 D. cyl. ax. 90° in each eye.

On examination December 3d, the corrected vision in the left eye was 20/12+, and in the right eye 20/40. The left eye was normal throughout. The right eye showed a small 0.2 D.D.-size, gray, hazy, disturbed area in and about the fovea. The surrounding retina was normal, the involved gray area apparently edematous. There was a right relative 3-degree central scotoma. On subsequent days the lesion became more gray, definite, and circumscribed. Investigation revealed a cloudy left antrum, four definite apical dental abscesses, chest findings by X ray suggestive of tuberculosis, and a positive tuberculin reaction.

The patient was hospitalized on December 6th, and 10 typhoid injections were given. Four abscessed teeth were removed. The gray edema subsided and there was present at the time of this report a regular round, slate-gray 0.25 D.D.-size area with a darkened red center. Vision at this time was 0.4+1 corrected. Tuberculin therapy was being continued.

Bilateral retinitis proliferans cum diabetes mellitus

Dr. R. I. Pritikin said that S. S., a woman aged 63 years, has been a known diabetic since 1928, and has been taking insulin since 1929. The diabetes had been supposedly under control. Failing vision was first noticed seven years ago. Despite a steady downward trend in visual acuity, she could read newspapers until about September, 1939.

Examination on April 6, 1940, showed corrected vision R.E., 2/200 and L.E., 20/200. The correction was —0.50 D. cyl. ax. 90° with +2.50 D. sph. add in each eye. This could not be improved. There was an extensive picture of bilateral retinitis proliferans, together with marked diabetic retinopathic changes. The proliferating gray-yellow mass with many new-formed vessels extended for 8 to 9 diopters into the vitreous of the right eye and from 1 D.D. above the disc to 2 D.D. nasally, 4 to 5 temporally, and 8 to 10 inferiorly. The disc was practically

invisible. The picture in the left eye was similar although to a lesser extent.

Baso-frontal syndrome

Dr. C. F. Schaub presented J. C., a white man aged 33 years, who had been shown to this clinical group about one year ago. At that time the case was presented as one of bilateral papilledema of 2 diopters with 20/12 corrected vision in each eye and peripheral-field findings (irregular constriction, mostly upper quadrants) in the left eye only. Neurologic examination, blood and spinal-fluid examinations, and air studies were all normal. There were no subjective symptoms except consciousness of the disturbed field of vision in the left eye. The sphenoid sinuses, which showed Roentgen-ray evidence of infection, were operated on in April, 1939.

Until November, 1939, a tentative diagnosis of nonspecific chiasmal arachnoiditis was made. The central vision, however, remained 20/20+ in each eye and the visual fields of the right eye were repeatedly normal. In view of this, plus the fact that the papilledema was slightly regressive, conservative therapy was felt justifiable. In November, 1939, there appeared signs of "whiteness" of the nerve head of the left eye, showing behind the still-present 1 to 1½ diopters of papilledema. The edema remained 1 to 1½ diopters in the right eye, the left visual field was further constricted, and the right visual field still normal. There were no subjective complaints, and neurologic examination, including careful investigation of the sense of smell, was normal.

In April, 1940, central vision was 20/30—4 and there was further loss of the peripheral field in the left eye with a relative central scotoma (early) and more evidence of postneuritic atrophy in a still-swollen nerve head. Vision in the right eye was 20/12 with a 1½-diopter papilledema still present. There were still no peripheral nor central field changes in the right eye.

Considering the possibility of multiple sclerosis, and so forth, it was felt that the clinical picture objectively was one of a baso-frontal syndrome. Exploration was advised. Consultation with Dr. Sanford Gifford, also with the neurosurgical and ophthalmological departments of the Mayo Clinic, confirmed the diagnosis. It was felt that either a baso-frontal neoplasm or an inflammatory lesion of the chiasm would be found. The maintained normal central vision and normal field of vision of the right eye was considered to be a point against an optico-chiasmatic arachnoiditis, while the absence of general subjective symptoms argued against neoplasm. However, the consensus of opinion was that surgical exploration was definitely indicated, lest the picture of atrophy appear also in the still-swollen right nerve head.

Central angiospastic retinopathy (left)

Dr. C. F. Schaub said that J. B., a white man aged 45 years, was first seen in April, 1940. He stated that his right eye had been practically blind since childhood and that it had always turned in.

Vision in his left eye was normal until April 11, 1940, when it suddenly became foggy. The attack reached its height in about 30 minutes. Wherever he looked there was a large black spot. There was no pain. He had consumed a minimum of a pint of whiskey daily and more than a quart on each Saturday and Sunday over a period of at least 10 years. He smoked two packages of cigarettes a day. His diet had been noticeably deficient in vegetables.

Examination showed vision R.E., 10/200 with +3.00 D. sph. ⊂⊃ +1.00 D. cyl. ax. 90°. This eye turned in 10 degrees with and without correction. The cornea, media, disc, and macula were

ophthalmoscopically normal. Vision L.E. was 1/200 (side glance—eccentric) with +3.00 D. sph. ⊂ +1.00 D. cyl. ax. 90°. Peripheral fields of vision were normal to form and color. There was a definite large cecocentral scotoma; cornea was normal; pupil responded sluggishly; disc was normal. The macula showed a disturbance in the form of a gray vague sheen in and above and below the fovea as compared to the rest of the fundus and to the normal right macula. The Gullstrand ophthalmoscope gave a more definite picture—the gray ischemia appeared in the deep retinal layers. A diagnosis of left central angiospastic retinopathy was made. Physical examination showed a blood pressure of 140/90; the blood count was normal and the blood Kahn test was negative.

Massive doses of vitamin B, both by mouth and injection, together with potassium iodide and amyl-nitrite inhalation were prescribed. There was little improvement and accordingly, on May 1st, the patient was hospitalized for a course of typhoid-vaccine therapy. Eight injections, up to 200 million typhoid organisms were given, together with pilocarpine diaphoresis and continuation of potassium iodide and vitamin B. Vision in the left eye improved from 5/200 on admission to approximately 20/40+2 with correction. While there was a central scotoma in the right eye, it was felt that this was not due to a macular angiospastic process, but rather to amblyopia ex anopsia since the macula of the right eye did not show the same ophthalmoscopic changes as did the left.

Syphilitic optico-chiasmatic arachnoiditis

Dr. C. F. Schaub said that J. V., a white man aged 34 years, first seen in October, 1939, had been receiving antisyphilitic therapy for two years. The time of primary infection was unknown. The vision had become progressively more impaired. On examination: vision, R.E., light perception in temporal quadrant only; L.E., 10/200 with a 3- to 5-degree absolute central scotoma. Pupils were irregularly fixed at approximately 5.0 mm. Media were clear and there was present a mixed type of atrophy (primary) much more advanced in the right eye. Peripheral fields of vision showed in the right eye a field only on the temporal side, obtained with a large 10-degree white object. The left visual field (4-mm. test object) gave a 3- to 5-degree definite absolute central scotoma and a definite 10- to 20-degree irregular constriction. No colors could be recognized in either eye. Blood and spinal Kahn tests showed 4+ reaction. Neurologic examination was negative. There had been a rapid loss of visual field, especially in the right eye. In spite of a course of fever therapy, there was a steady downward trend, and chiasmal exploration was advised and performed on December 28, 1939, by Dr. H. C. Voris. The usual picture of plastic optico-chiasmatic arachnoiditis was found. Adhesions binding nerves and chiasm were freed and removed. On last examination, April 20, 1940, the vision of the right eye was light perception in the lower temporal quadrant only, which remained stationary. The vision in the left eye was 10/200; the central scotoma persisted. The peripheral field to 3-mm. white test object was definitely improved, and there was definite perception of blue color and a fair blue field.

The case was presented because of the quite typical findings of syphilitic optico-chiasmatic arachnoiditis syndrome picture, which despite vigorous antisyphilitic therapy was becoming rapidly worse.

Compensated simple chronic glaucoma, homonymous hemianopsia

Dr. Weem said that F. S., a woman aged 64 years, was first seen in August,

1937, when she was referred from the diabetic department.

The uncorrected vision at that time was R.E., 20/30−1; L.E., 20/40+2, vision with glasses 20/20 each eye. The intraocular tension was 18 mm. Hg as taken with the Schiötz tonometer.

Fundus examination showed moderate retinal angiosclerosis. The retinal veins were moderately full. There were a few scattered grayish-white exudates in the right nasal mid-periphery. A number of punctate and irregular hemorrhages were noted in each posterior polar region. There were also a few fine miliary aneurysms.

In May, 1938, the patient was treated for acute conjunctivitis. In December of the same year, she was admitted to the dispensary as an emergency case, complaining of terrible dizziness for a few days. No rainbow colors were noted. The corrected vision of each eye was 20/50. The intraocular tension was 31 mm. Hg in each eye. Visual fields were taken, and revealed a left homonymous hemianopsia. Eserine salicylate, 0.3 percent, was instilled while the patient was in the dispensary, and a prescription for pilocarpine, 2 percent, to be instilled three times daily was given. Within the next 24 hours, the intraocular tension dropped to 18 mm. Hg in each eye. The patient was admitted to the hospital on the neurological service, and the final diagnosis was cerebral arteriosclerosis.

The patient remained under constant observation in the out-patient department. The tension fluctuated between 16 and 21 mm. Hg as long as she continued to use pilocarpine, but went up to 30 mm. Hg immediately when the drops were discontinued. The vision was 20/20 in each eye. Repeated tests of the visual fields showed no changes in the existing hemianopsia. The maximum blood pressure was 175/95.

Globular posterior vitreous detachment

Dr. Paul Hurwitz presented a white woman, aged 33 years, who complained of moving spots in front of the left eye.

Vision: R.E., 1.5; L.E., 1.5.

The left eye showed several scattered round pigment deposits on the posterior cornea and numerous coarse vitreous floaters. In the superior temporal periphery there was an area of old chorioretinitis, 3 D.D. in size with massive pigment proliferation. Clearly visible with a +8.00 D. sph., anterior to the disc, was a posterior central detachment of the vitreous, which was seen as a freely floating ring-shaped opacity with bright reflexes that assumed an oval or slitlike appearance. Fundus details were hazy elsewhere, but were clearly visible through the center of the ring (window phenomenon).

Complete physical examination revealed an erosion of the cervix and periapical infection of all the lower teeth. Other findings were negative.

Bilateral incipient cataracts—bilateral Kuhnt-Junius choroiditis

Dr. David Holden said that J. R., a white man aged 79 years, complained of gradual loss of vision for six years. Corrected vision in each eye was 20/200. He was able to read large type with +13. D. sph. Peripheral fields were constricted. There was a central scotoma for form and color. Tension: R.E., 10 mm. Hg; L.E., 10 mm. Hg.

Medical, laboratory, and X-ray findings were normal. The right lens showed peripheral spokes between the 3- and 6-o'clock positions. The left lens showed peripheral spokes between the 6- and 12-o'clock positions. Both maculae were 2 D.D. in size and showed grayish, irregular areas, elevated 1 diopter, which were irregularly pigmented at borders.

Lesion of undetermined origin of the right macula

Dr. David Holden said that A. S., a white woman aged 35 years, had had frontal headaches for one year. Vision R.E., 20/65; with +0.50 D. sph. ⊂⊃ +1.00 D. cyl. ax. 30° it was 20/30. Vision L.E., 20/50; with +0.75 D. sph. ⊂⊃ −2.75 D. cyl. ax. 30° it was 20/50. Tension R.E., 15.5 mm. Hg; L.E., 15.5 mm. Hg.

Peripheral and central fields were normal. Medical consultation and laboratory findings were negative. X ray showed all sinuses hazy, the frontal undeveloped. The nasal turbinates were hypertrophic. The fundus of the left eye was normal. The macula of the right eye showed a deeply reddish oval area one-seventh disc diameter in size with radiating streaks extending temporally.

Clinical studies concerning the alleged synergistic role of benzedrine and paredrine in homatropine cycloplegia

Dr. Karl J. Scheribel presented this paper in which Dr. William F. Moncreiff was collaborator, and which has been published in this Journal (March, 1941).

Discussion. Dr. J. E. Lebensohn said that this was the most thorough and best-controlled paper on this subject that he had heard. He objected to the intimation that these sympatheticomimetic drugs have any paralyzing effect on the accommodation. The apparent loss of 3 to 5 diopters of accommodation can be explained by the pupillary dilatation. This could have been checked by making the tests with an artificial pupil of a size equal to that previously present when the accommodation and convergence were normally active.

Dr. William F. Moncreiff congratulated Dr. Scheribel on his excellent presentation of the paper. He said that misleading statements have been made on this subject by other authors—one is that maximal dilatation of the pupil is, other things being equal, the most desirable condition for the examination of the refraction, and, since there is considerable evidence that the use of Benzedrine or paredrine in addition to homatropine gives a wider pupil than the same dosage of homatropine alone, this is a point of advantage for the use of these drugs. The fact is that the contrary is true. A fixed pupil of 5 to 6 mm. is more desirable than one that is wider, both for the subjective and objective (skiascopic) tests, as every competent refractionist knows.

The second point concerns intraocular tension. Some authors have stated that benzedrine and paredrine are less likely to induce elevation of the intraocular tension, with dilatation of the pupil. However, a clear-cut distinction must be drawn between the glaucomatous and the nonglaucomatous eye. The use of any mydriatic in a glaucomatous eye of any type may be productive of a further rise of tension, or even of an acute congestive attack. The extent of this hazard depends, on the one hand, upon the type, stage, or severity of the glaucomatous process, and on the other hand, on the dosage, relative power of action, and duration of action of the mydriatic. In this regard, benzedrine and paredrine are not by any means innocuous, nor are they less dangerous than other mydriatics of similar power and duration of action. In the nonglaucomatous eye, all mydriatics fail to produce a definitive rise of intraocular tension, regardless of maximal mydriasis. The slight variations of intraocular tension, in a pharmacologic sense, attendant upon the use of this or that mydriatic in the normal eye, are not of the slightest clinical importance.

Relative to the apparent partial cycloplegia with the use of paredrine alone,

in this small group of nine patients, all under 21 years of age, and with accommodative powers ranging from 10 to 13 diopters, it may be that the dilatation of the pupil, as suggested by Dr. Lebensohn, was an adequate explanation.

Robert von der Heydt.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

May 6, 1940

Dr. Arthur M. Yudkin, *presiding*

Symposium on ocular tuberculosis

Pathology of ocular tuberculosis

Dr. Louise Meeker discussed this subject during the instructional hour. Slides of ocular tuberculosis and other conditions presenting a similar picture were shown for differential diagnosis.

Demonstration of experimental ocular tuberculosis in rabbits

Dr. Ernst Metzger read a paper on this subject.

The influence of allergy on some clinical pictures of ocular tuberculosis

Dr. Joseph Igersheimer stated that some bacteriologists including Calmette believed that general tuberculosis is often due to bacilli entering through the conjunctiva. Wherever a tuberculous infection takes place in a normal body the regional glands are soon affected, and this process is often more pronounced than the primary lesion. If the same infection takes place in an already tuberculous patient the regional glands do not react at all or very little. This fact is proved by two series of cases of conjunctival tuberculosis.

In the first group of 40 cases, in which there was a real abscess of the preauricular glands, no signs of general tuberculosis existed. In a second group of 54 cases, with no signs of regional-gland involvement, other manifestations of general tuberculosis always existed in the lungs, pharynx, and elsewhere. Whereas in the first group the tuberculosis of the conjunctiva was due to an ectogenous infection, the source of the infection in the second group was uncertain.

Primary intraocular tuberculosis is so rare and hard to prove that we always speak of a hematogenous secondary infection in these cases. Bacilli entering the globe encounter the more or less allergic tissues of the uvea and other membranes. Since we cannot determine the allergic state of the ocular tissues themselves we have to resort to the general skin test. A lack of correspondence between the intensity of the eye disease and the skin-test reaction may be due to the varying sensitivity of skin and uvea to tuberculosis toxin. The skin test may be more important from the allergic standpoint than the diagnostic, although cases of histologically proved ocular tuberculosis with negative skin tests have occurred.

Clinically ocular tuberculosis may be of the exudative or proliferative type. The former is characteristically an acute though nonspecific inflammatory reaction caused by tuberculoprotein which always occurs when protein (as antigen) and antibody meet. The proliferative reaction is the formation of specific tuberculous tissue and is due to the tubercle bacillus. In cases of high uveal allergy it is probable that acute, intense, inflammatory tuberculosis may occur. Such cases are found in the literature.

Other acute tuberculous processes are the miliary tubercles. Here allergy and resistance are low, the patient has a severe tuberculosis, and the disease is not produced by a high allergy but by a mass of

bacilli in the blood stream. A rare characteristic condition is the conglomerate form of tuberculosis, especially of the anterior part of the globe. This is especially common in children and may be explained by the fact that these patients are in a very early stage of their general tuberculosis in which the allergy is often high, according to Ranke, and the immunity-resistance low. But the number of bacilli may also be very important.

It can be accepted as a fact that a real phthisis produces immunologically a high degree of resistance to Koch's bacillus in the other body tissues, and it has been shown that with a severe pulmonary tuberculosis an ocular process almost never occurs.

Discussion. Dr. Percy Fridenberg asked for one or two more definite suggestions as to the differential diagnosis between the allergic and bacterial types of lesions, and whether there is any choice of treatment in these two forms. The antagonism is an interesting point. He asked if there are any data as to the relation of ocular with systemic, nonthoracic tuberculosis, saying it would be interesting if a connection could be shown.

Dr. Sigmund A. Agatston said that Dr. Igersheimer's findings agreed with his. He felt that an ophthalmoscopic distinction could be made between the allergic and proliferative types. Clinically the inflammatory type usually shows numerous vitreous opacities with hemorrhages so that the lesion cannot be seen clearly. In the proliferative type there are but few vitreous opacities. He added that he had examined a large number of tuberculous patients in a sanatorium and was impressed with the fact that he rarely saw a tuberculous choroiditis in a patient with an active tuberculosis.

Dr. Joseph Igersheimer in closing said that a diagnosis of tuberculosis of whatever type is usually a diagnosis of probability. Of course, cases of conglomerate tubercles in children are certainly tuberculous. In adults, even with nodules in the iris, one is not so certain. He believed that tuberculous infection is very often the cause of uveitis, although the picture is often different. Even histologically there is sometimes nothing specific in the uvea. There is an inflammatory process, but no nodules, no epithelial cells, no giant cells. In the future we may know more about this.

As for the treatment, a few words should be said about tuberculin. When we believe that the reaction is one of allergy in which tuberculin is present in the tissues or transported from one place to another by the blood, the adding of more tuberculin should increase this reaction. On the other hand, if we can bring this tuberculin to the focus without increasing the inflammation too much, then we desensitize the allergy. The tuberculin produces antibodies and in such cases where there is great hypersensitivity we can decrease the degree of allergy by tuberculin. There is danger in this focal therapy because if the dose is too large (and we do not know how to judge it) there may be a focal reaction and an increase of the whole disease. But even in these very hypersensitive cases if one is very careful and uses small doses the therapeutic effect may be good. Radium has been of help in some cases but not in others.

Irradiation treatment of ocular tuberculosis

Dr. Ludwig Sallmann said that sunlight is the most important part of general irradiation therapy. The experiences of Werdenberg, Rossler, and Wegner showed that this, combined with other features of mountain cure, did succeed in serious forms of uveal tuberculosis if continued long enough. Lupus of the lids is probably best treated by thermocoagu-

lation followed by irradiation in small doses. Most radiologists agree that radium is superior to X rays in the treatment of most forms of conjunctival tuberculosis.

Tuberculosis of the tear sac and gland can only be attacked by the penetrating rays such as the gamma rays. Tuberculous keratitis and scleritis have been reported cured by ultraviolet, border, radium, and X rays. Their advantage over other forms of conservative treatment, however, is not definitely proved.

X-ray and radium therapy seems to be of definite value in preventing serious relapses. Focal and general irradiation is an important auxiliary treatment of tuberculosis of the eye. This may be applied without damage to the other structures if the technical advice of radiologists is carefully followed.

Discussion. Dr. James W. Smith stated that Dr. Woods of Baltimore is opposed to altitude therapy, and he finds that the resistance of the patient is built up by modern recognized methods including rest, sunshine, and vitamins, augmented by tuberculin therapy.

Dr. Ralph I. Lloyd said that he would like to emphasize the value of rest in the treatment of tuberculosis of the eye. He felt that its value was underestimated and that he had considerable faith in the modern forms of fish-liver extracts.

Dr. Joseph Igersheimer said that Werdenberg rarely uses tuberculin but used sunshine, climate, and other nonspecific therapy. He said he was afraid of tuberculin but this was probably not justified. When tuberculin is used the bacilli themselves are not affected but the toxic substances are desensitized and produce a better surrounding and increase the resistance of the body. Climate, vitamins, and the like are intended for the same purpose.

Dr. Ludwig Sallmann in closing said he could not explain the difference in findings between Woods, Werdenberg, and others. He was convinced of the value of heliotherapy and altitudinal therapy by results he saw in Greece and Germany. While he did not advise radium and X-ray therapy for all choroidal lesions he believed that the proliferative lesions often responded remarkably by a disappearance of exudate and nodules.

SIDNEY A. FOX,
Secretary.

ROYAL SOCIETY OF MEDICINE

SECTION OF OPHTHALMOLOGY

June 14, 1940

MR. MALCOLM HEPBURN, *chairman*

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CATARACT IN ADULT RICKETS (OSTEOMALACIA)

MR. J. PRESTON MAXWELL said that at the time the paper on "Adult rickets" was delivered to the Royal Society of Medicine in 1934 they were already aware of certain degenerative processes which are occasionally seen in connection with osteomalacia. Pi, in 1934, took up the study of cataract in relation to their osteomalacia cases, with relation to what goes by the name of "cataracta tetanica." Cataract has never been especially associated with osteomalacia, which makes the subject an important one, as it is clear that the term "cataracta tetanica" is, in some cases at least, a misnomer.

One has to look especially for this form of cataract, as the subjective ocular symtoms are slight; and as the majority of their patients are illiterate, it is only when the disease has considerably advanced that the patients are likely to complain of loss of vision.

Out of 60 well-marked cases Pi found 13 cases of this form of cataract, making an incidence of 21.6 percent.

The numbers are of course small, but the probable incidence in marked cases is at least 15 percent.

Cataracta tetanica was first described in 1872 by Logetschnikow. He reported 14 cases in which spasms were present at the time of examination or a history of convulsions preceding the disturbance of vision was obtained. The same trouble was also observed by Schmidt-Rimpler (1883), Bernhardt (1891), Wettendorfer (1897), and others. These cataract cases were mostly in young or middle-aged women, none of whom was stated to have been suffering from osteomalacia, though it is quite possible that some were so suffering. Bernhardt's observation first led him to use the term "Cataracta in idiopathic tetany" for the kind of cataract in which the nature of the tetany was not known.

Landsberg (1888) later recognized and reported cataracts in postoperative tetany; that is, the cataracts developed after accidental removal of the parathyroids or were due to their degeneration after thyroidectomy. Erdheim (1906) and others have also described these in experimental work. Cole (1930), in giving an account of a typical human case, cited descriptions of cases by Jacques, Saintor and Peron, Aub and Hunter.

So it is clear that the disease can be classified into cataract in postoperative cases, and cataract in idiopathic tetany. According to present knowledge both of them are associated with disturbance of endocrine secretion, the only difference between them being that the development of cataract in the former has a known cause; that is it results from the removal of the parathyroid glands, while the etiology of the latter has not yet been clearly explained. Similar symptoms such as changes of hair and finger nails, caries of teeth, and diminished activity of bone growth have been observed in both kinds of affections. Tetany is common in osteomalacia, and where a history of tetany cannot be obtained there has often been numbness of hands and feet.

A review of the literature reveals that the cataracts in association with idiopathic tetany are reported as follows: (1) lenticular opacities as one of the complications in pregnant women with tetany by Meinert (1887) and many others; (2) pregnancy in rachitic women complicated by tetany cataract, Janacek (1920); (3) cataracts in a rachitic woman suffering from convulsions. All of these are possibly of the same type as those they discussed and details of which were given in tables.

It will be noted that in some cases the blood-calcium is only slightly below normal, while the blood phosphorus is extremely low. The density of the lenticular opacities varies considerably. In its early stage it is always mild. In these cases it can be detected only by means of the slit-lamp. It is composed of very fine, dust-like opacities in the form of an opaque zone situated right beneath the capsule of the lens. As the disease progresses, the opacities get bigger and extend towards the cortex, with at the same time some water slits. The diagnosis of cataract can then be made with the naked eye. In the course of time the cataract will undoubtedly become ripe, although it runs a slow course, as is evidenced by two cases which came to the eye service direct. It is therefore obvious that any middle-aged or young woman in whom this form of cataract is found should be carefully examined for adult rickets before a diagnosis of complicated or presenile cataract is made. Under appropriate treatment these cataracts undoubtedly improve, but do not disappear. To date there is no medical therapy for cataract.

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LEARNING OPHTHALMOLOGY

The real student does not study for good marks, but to gratify his inward desire and impulse to expand his knowledge and develop his powers of thought. He enjoys thinking. The best education is thus gained. An experience of complete diphtheritic paralysis of accommodation is a good preparation for the study of refraction. It makes you interested in it. Medical students have kept notes of the lectures they have heard. Some have amplified their notes and carefully written them out for subsequent reference.

If these notes were laid aside and not read afterwards they soon lost their original significance and suggestive value. Those who have practiced note-taking as students may continue their course of study which had only begun when they received the title of M.D. They can go on by taking records of cases in which they become really interested or which seem especially important. The doctor who has such case records and turns to them to refresh his memory when similar cases are encountered has made an important beginning in graduate study. Note-taking and study of the notes, by the light of what has been subsequently learned, are valuable as a means of keeping in touch with one's own experience, ripening his judgment, and getting a broader view of the branch of medical practice to which he has devoted himself.

One who determines to succeed and

take a leading place in any branch of modern science must pledge himself to a lifetime of continued study. Medical societies had become study clubs, by their reports and discussions of cases and of new methods, before colleges and universities realized that all medical men need opportunities and facilities for graduate study. Now the institution that most fully realizes this will best hold the interest and support of its graduates and the medical profession.

Active and important medical societies, like state medical societies and those devoted to special branches of practice, generally publish annual transactions. These volumes may be referred to by those who have them, and may be read by younger members who hope to contribute to such transactions in future. But the periodical transactions of medical societies are not the most important class of medical literature.

At the last annual Mid-Winter Clinical Course of the Research Study Club of Los Angeles, Dr. H. G. Merrill, of San Diego, called attention to the eye notes he had written himself, or had taken by a stenographer, at all the preceding courses. These notes had been revised by himself and some of them had been revised by those who had given the lectures. They had all been carefully condensed. He offered them for publication if enough copies were subscribed for to pay the cost of printing.

This is a new kind of medical literature. It may set an example of intensive study and careful review to any conscientious student. If he has notes of his own, supplemented by his reading, memories, and impressions made by striking cases, he has a basis and inspiration valuable to any student of medical science. The ophthalmologist who attends courses on otolaryngology may not be planning to increase his practice in that direction. It may mean that in those courses he sees medical science and practice from a different point of view, and really gets a broader view of the general field of medicine and surgery. When the ophthalmologist hears lectures on diseases of the upper respiratory tract, he can feel that he is keeping in touch with problems of infection, general nutrition, and the climatic influences. He is really keeping up an interest in recent medical progress.

Edward Jackson.

ISEIKONIC ENTHUSIASM

Therapeutic nihilism is the opposite of therapeutic optimism. The latter may tend to swallow too eagerly claims made as to wonders wrought by a new remedy. The former is the attitude of the skeptic who insists upon very complete proof before admitting that he is convinced.

Osler was sometimes accused of being a therapeutic nihilist, because he wisely recognized the relatively insignificant part played by drugs in the cure of disease.

"A lens," once said a leading optometrist," is not a pill." But the results obtained from the prescribing of correcting lenses were for a time subject to the same tussle between optimist and nihilist as have been some products of the pharmaceutical laboratory. A few leaders in medicine were at first ready to sneer at the claims of the ophthalmologist as to the relief of gastric and neurologic symptoms by the correction of refractive errors. On the other hand, ophthalmologic claims now and then displayed credulity on the part of physician and patient.

George M. Gould, who loved controversy and never hesitated to say what he thought, had a very complete faith in the blessings conferred by the expert refractionist. He loved to trace in the lives of the great, in literature, art, music, and

other walks, evidence of incapacity due to need for proper glasses.

But Gould, whose own technique in refraction, though meticulous, was occasionally marred by inaccurate thinking, made a number of statements which suggested overenthusiasm. He was disposed to attribute to eyestrain the group of symptoms usually labelled as belonging to Ménière's disease. He was a little too ready to regard as authentic all the claims made as to the cure of epilepsy by glasses. He blamed upon eyestrain almost all cases of migraine instead of recognizing that migraine might arise from widely different causes. Among the other calamities which he blamed upon uncorrected refractive errors were choreic movements, heart pains, extreme depression, spinal curvature, insomnia, and suicide.

There can be little doubt that Gould was right more often than wrong. He was certainly a public benefactor, both in his practice of ophthalmology and in his medical writings. Yet it is likely that his tendency to dogmatic overstatement sometimes damaged the cause which he championed.

Several ophthalmologic causes have had their too passionate advocates. Take, for example, the earlier craze for fitting prisms, or even for some now less popular types of muscle surgery. Rightly or wrongly, there are today some excellent ophthalmologists who believe that we are going through a similar phase of exaggerated faith and enthusiasm as regards the permanent results to be obtained from orthoptic training.

The new "profession" of optometry, not yet authorized by law to employ drugs in the care of the eye, adopts with enthusiasm any mode of treatment, particularly optical or mechanical, which does not conflict with the restrictions of the law as to the practice of medicine. Hence the optometric vogue for certain uses of prisms and ocular "exercises" which were temporarily picked up and then for the most part dropped very promptly by our own profession many years ago.

In considering any new therapeutic procedure, however, we must not forget that the credit for discovery of principle or method is not diminished by the fact that the scientific discoverer is not a medical man. The observations of Airy, astronomer, and Young, physicist, on astigmatism were just as genuine and valuable as though they had been made by members of the medical fraternity.

The lead, in this country at least, with regard to troubles arising from differences in size of cerebral image has come from scientific workers who are not physicians; and it may be that this fact has somewhat retarded medical acceptance of the principles enunciated by Ames and his associates. Any such prejudice is undesirable.

More important, in retarding widespread investigation of technique and results, have been the expense and the mechanical difficulties associated with the necessary apparatus.

A further difficulty arises from the conviction that conclusions as to the effect of size differences in the cerebral image can only be thoroughly reliable when the accuracy of basic refractive measurement of a patient is beyond doubt. But it is unfortunately true that we lack standardization in refractive technique. When a physician is called upon to refer a patient to an ophthalmologist in another city, there is no detail in which he is likely to experience greater uncertainty than as to the refractive methods employed by the distant colleague, for the best-known eye surgeon may be occasionally a much less capable refractionist.

The subject of aniseikonia is worthy of fuller investigation in various parts of the United States; and the Dartmouth Eye

Institute is evidently taking steps to promote such local study of the problem. But it is to be hoped that the enthusiasm of these clinical centers will not lead them to discredit their field of labor by indiscriminate publication of claims which many excellent ophthalmologists may consider exaggerated, and which may create misleading expectations among the laity.

A circular from one of these new centers presents an elaborate table of "symptoms indicative of aniseikonia," under the headings of "functional," "local," "neurologic," and "gastro-intestinal." The list reads remarkably like a classical recital of symptoms attributable to eyestrain.

The functional symptoms listed are: asthenopia, photophobia, car or train sickness, excessive fatigue in reading or working at near range, discomfort in attending movies, lectures, etc., and impaired fusion and stereopsis.

The local symptoms: eyes burn or ache, blurring, diplopia, lacrimation, pulling sensation, and blepharospasm.

The neurologic symptoms: headaches, vertigo, psychoneurosis, general fatigue, tension or irritability, general nervous fatigue, feeling of pressure in the head, and pain or tenseness in the cervical region.

The gastro-intestinal symptoms: nausea, indigestion, and gastric disturbances.

Since the difference in size of images is presumed to reside in the brain, and can hardly therefore make important demands upon the nerve or blood supply of the eye or its external adnexa, one wonders how such symptoms as photophobia, burning or itching of the eyes, blurring of vision, lacrimation, and blepharospasm can arise from this cause.

The "aniseikonia bulletin" from which this list is quoted, issued by the Aniseikonic Clinic of a well-known western "sanatorium and hospital," carries in its heading the name of an ophthalmologist M.D., and that of an "O.D." (presumably Doctor of Optometry*). In an announcement card accompanying the bulletin it is stated that the work will be done with the instruments and methods indorsed by the Dartmouth Eye Institute and that it will be in charge of Dr. —— (the ophthalmologist), and of "Dr."* —— (the optometrist) as technician.

W. H. Crisp.

GONIOSCOPY IN GLAUCOMA

The classification of glaucoma has long been a confused one and should be clarified. It has been described as being divided into two principal types, the primary and secondary. Primary glaucoma has been subdivided into such categories as acute congestive, chronic congestive, and chronic noncongestive or simple glaucoma. Another classification divides the types of glaucoma into compensated and incompensated. It is rather generally conceded that reclassification is necessary but, as Gradle asks, "do we know enough yet to make it?" Raeder in 1923 introduced a classification based on chamber depth. The opinions were the result of clinical observation.

Recently, gonioscopy has made great advances, and it would appear that further study of the angle of the anterior chamber may give us a better understanding of the etiology of the disease and through this a more rational classification.

With a better understanding of the disease the therapy can be handled more intelligently.

In 1933 M. Uribe Troncoso and A. B. Reese presented a paper before the Oxford Ophthalmological Congress on go-

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* At both these points the Aniseikonic Clinic seems to be at variance with the law of the state, which apparently denies to optometrists the professional use of the title "Dr."

nioscopic findings following the Elliot operation. They stated that the examination of the angle of the living eye by means of the gonioscope and contact glass has passed from the "era of experiment to the stage of clinical application." In their opinion this examination also gave important information in a variety of conditions related to the angle of the chamber of the living eye. They advocated that with this method every ophthalmologist, after some training and with a little patience, should be able to "see the angle as easily as the anterior segment with the slitlamp." This presupposed that the ophthalmologist was properly trained in the use of the gonioscope and in the anatomy and physiology of the region. To the average ophthalmologist, however, the procedure seemed too complicated to warrant its adoption in practice.

Stimulated particularly by the work of Troncoso, Otto Barkan in 1936 developed his method of gonioscopy, using the microscope head of the slitlamp suspended from an easily adjustable arm. This method has greatly simplified the procedure, so that an examination can be made in the office in a very few moments.

Since Troncoso's work was published, various papers have appeared from time to time on the findings in the angle in glaucoma as seen by the gonioscope. That this method of examination may become of importance in the intelligent care of glaucoma is suggested by the recent appearance in the American Journal of Ophthalmology of two rather practical papers on this subject.

In Otto Barkan's paper, read before the Pacific Coast Oto-Ophthalmological Society in 1939, he suggests that the choice of operation in glaucoma be based on its classification according to the following types: "trabecular glaucoma with a wide or open angle or a narrow angle, or iris-block glaucoma with a shallow chamber and narrow angle." This classification was based on work done since 1936.

This year Sugar read a paper before the Chicago Ophthalmological Society in which he also reports his theory and classification based on work done by Gradle and himself during the last few years. Their findings in general corroborate the findings of Barkan.

The continued study of the iris angle by means of gonioscopy in the various ophthalmological centers of the country should eventually give us a better understanding of the etiology of one of the most difficult diseases with which the ophthalmologist must cope.

The work done so far would suggest that the type of glaucoma can be determined by study of the angle, and that the operative procedure indicated may be based on gonioscopic findings rather than on purely clinical judgment. If this should be true it would probably greatly improve the operative prognosis of glaucoma.

We have all had the experience of carefully controlling the tension medically and having the patient disappear to return later with complete loss of vision because he hadn't used his drops. Often he states that he went away from home for a time and forgot his drops and found that the eye didn't bother him so he felt the drops were no longer necessary. This occurs with sufficient frequency to cause some ophthalmologists to feel that every case of glaucoma should be classed as surgical.

With a better understanding of the disease and a guide with which to decide upon the operative procedure, we may be able to improve markedly the surgical results. If this can be accomplished, perhaps the therapy of glaucoma may in the future be regarded as purely surgical.

Ophthalmologists should read all papers on the study of glaucoma with the

gonioscope with an open mind, and more detailed studies should be made of the disease with this newer method. As a result of this we may completely revise our conception of the etiology and care of glaucoma.

Frederick C. Cordes.

VITAMIN THERAPY IN OPHTHALMOLOGY

Elsewhere in this issue are to be found two articles, by careful investigators, on eye diseases due to vitamin deficiency and their treatment with vitamins of various sorts. It is unnecessary here to recall the history of the discovery of the vitamins and the disorders produced by the lack of them in both the animal and man. The subject forms a glorious and fascinating current chapter in medicine, and every physician is now awake to this information, if not from scientific sources, at least from the literature of the drug houses and from the daily press and lay magazines. One can say that there is no deficiency of vitamin awareness, at any rate.

It is but natural that in the enthusiastic reception of this knowledge, abuses should have crept in. If the history of any drug or healing agent be investigated, this same abuse at the onset of its application will be found. A search of the medical literature of the past will disclose serious articles in first-rate journals by outstanding men, devoted to astonishing reports of successful therapeutic results in all sorts of conditions from the use of such drugs as quinine, thyroid extract, cocaine, and a host of others including tobacco. The first flush of excitement following the announcement of a therapeutic discovery led naturally to its application to a legion of misunderstood conditions, sometimes with an amazingly good effect; witness, for example, the value of sulfanilamide in trachoma, often with no effect and occasionally with disastrous and unanticipated results, as, for example, the cataract formed consequent to the dinitrophenol treatment of obesity.

Our knowledge and application of vitamin therapy is at present in the state of ferment and flux through which the evaluation of all of the drugs in the pharmacopeia have passed. An enthusiastic hunter sees an Indian behind every tree. Any patient with an ocular condition that has so far defied an exact knowledge of its treatment is now being dosed with vitamins that are given locally and generally in shotgun mixtures. They have been fanatically prescribed in conditions as far apart as cataract and retinitis pigmentosa, myopia and interstitial keratitis, primary optic atrophy and Mooren's ulcer. For example, unsupported claims are made daily, at least verbally, that cataract can be prevented, a fact most difficult to prove, and not only that, but cured as well by the ingestion of vitamins, either in capsule form under all sorts of confusing trade names, or by the daily consumption of one raw carrot. It is natural that the vitamin craze is wide open to exploitation by the unscrupulous. The only difficulty seems to be that the public is now so well educated that the physician finds that the majority of his patients are already taking vitamins of their own initiative and have been doing so for some time.

The only way in which our prejudices can be debunked is by the publication of articles by competent and trustworthy observers who have access to a large amount of clinical and experimental facilities. The question, "Is this honest work?" must be constantly posed. Subtle influences such as the desire to satisfy enthusiastic ambition, or to promote the interests of pharmaceutical benefactors must be searched for and weighed in the

balance by the reader. The logic of the drug action must be apparent to some extent at any rate. Finally, the test of one's own experience must be made and impressions turned into facts by observation.

The Journal will always welcome honest work that will help to solve this and all other ophthalmic problems. Even if later knowledge should disprove ardent claims, the forum will not have been in vain.

Derrick Vail.

BOOK NOTICES

PERSONAL HYGIENE APPLIED. By Jesse Feiring Williams, M.D., Sc.D. Seventh edition. 165 illustrations, 527 pages. Philadelphia, W. B. Saunders Company, 1941. Price $2.50.

The ophthalmologist will be interested to know that the era of preventive medicine is being appreciated by other branches of the medical profession. This author is the professor of physical education in the Teachers' College of Columbia University. The fact that the work has reached the seventh edition shows that educated people outside the medical profession begin to appreciate the value of prevention. The work is divided into 16 chapters, of which one is devoted to the Hygiene of the mouth, eye, and ear. In the seven pages devoted to the eye, the distinction is made clear between the eye physician and the optometrist. The importance of good light and the ways to secure it are emphasized, and the dependence of general symptoms on eyestrain is brought out.

This book is not intended to emphasize and explain the importance of good vision, and especial need for care of the eyes, but it does bring out the importance of personal health, and specify the many directions in which it may be developed and maintained; and the dangers that threaten its impairment in many directions. While it is addressed, and has an appeal, to all educated and thinking persons, it will be most generally appreciated by members of the medical profession. They will best understand the facts on which it is based, and the value of the health that it is possible for most persons to attain. For doctors who are engaged in special branches of medical practice, it will renew an understanding of the breadth of the field in which those who work for health must observe and exercise their professional skill.

Edward Jackson.

EL TRACOMA, REBELDE Y MILENÁRIO. (Trachoma, stubborn and ancient.) By Antonio Ros. 173 pages without illustrations, diagrams, or tables. Published by Editorial Cultura, Mexico, D. F., 1941.

This monograph on trachoma comprises a summary review of the subject with about half the text devoted to the historical development of the disease. This section is the most detailed part of the book and maintains an interest throughout its telling. The section on the clinical phases is brief and consequently lacks both sufficient detail and critical comment. Starting, for example, with adoption of MacCallan's classification, the etiology is then summarily presented with the Halberstadter-Prowazek body accepted as the incitant, and ending with a compilation of the surgical and medical methods of treatment. The author recommends for the management of trachoma the use of aspirin. The drug is used topically in the form of drops (0.03 gm. dissolved in 15 gm. of sterile distilled water), or it may be swabbed or pencilled on the conjunctiva as a solution in glycerine (0.3 gm. in 30 gm. of neutral glycerine). It

struck this reviewer as odd how little is said regarding sulfonamide treatment, since, irrespective of its actual or assumed specificity, the question is currently too important to be ignored. The final section of the monograph is devoted to the geographical distribution of the disease with statistics on blindness, in most cases including blindness due to trachoma. The final chapter presents a résumé of the more popular method of prevention.

The monograph ends with a bibliography of 295 references or 12 pages: 22 citations are of the *loco citato* variety, 13 are from the Bible, and only 69 represent communications published since 1930, the latest of which do not go beyond 1938, and oddly enough with scarcely a mention of the American investigations. It seems to this reviewer that the monograph in attaining brevity and simplicity simultaneously sustains lack of detail and omission.

L. A. Julianelle.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP
ASSISTED BY DR. GEORGE A. FILMER

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

10

RETINA AND VITREOUS

Paula Santos, B. **Angioid streaks of the retina.** Arquivos Brasileiros de Oft., 1941, v. 4, June, pp. 136-139.

A Brazilian workman aged 23 years was brought to the hospital during a spell of unconsciousness, perhaps induced by alcoholism. The incidental examination of his eyegrounds showed, in addition to some deep retinal hemorrhages, the typical appearance of angioid striae of the retina. From a single, almost complete pigment ring around the disc radiated many striae, of dark gray color, giving the general effect of tortuous vessels with branches. They were behind the retinal vessels, diminished in caliber toward the equator (beyond which they did not pass), and were accompanied by the usual retinal reflexes.

The dermatologic examination showed absence of elastic pseudoxanthoin, a fact referred to by the author as important since this skin affection commonly accompanies angioid streaks of the retina. The Wassermann test was negative although the patient stated that it had been positive some time previously and no treatment had been taken. Massive treatment with vitamin C was undertaken without benefit. (2 retinal photographs.)

W. H. Crisp.

Rauh, Walter. **The color field in experimental night blindness.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 545-548. (See Section 3, Physiologic optics, refraction, and color vision.)

Riddell, W. J. B. **Two clinical tests for night blindness.** Trans. Ophth. Soc. United Kingdom, 1940, v. 60, p. 181.

The visual acuity of patients with night blindness was definitely reduced as compared to that of normal individuals when taken in the light of a standard candle (of sperm wax and burning 120 grains of wax in an hour).

When the color vision was tested by means of Ishihara charts, all the night-blind patients named the colors correctly, but when tested with the Still-

ing charts these patients all made the same mistakes in the field of yellow-blue sensitivity. (Illustrations and charts.) Beulah Cushman.

Sullivan, G. L. **Embolism of the superior temporal retinal artery.** Amer. Jour. Ophth., 1941, v. 24, Aug., pp. 936-938.

Torres Estrada, A. **Detachment of the vitreous.** Bol. del Hosp. Oft. de Ntra. Sra. de la Luz, 1940, v. 1, Sept.-Dec., pp. 148-157.

The patient, a woman of 47 years, had suffered twenty years previously from bilateral iridocyclitis which led to pupillary seclusion. For this condition an iridectomy had been performed on each eye 18 years previously. In 1936 the patient came with cataract in each eye. Extraction was undertaken on the right eye two months later. The limbal incision was followed immediately by escape of a considerable quantity of yellowish fluid vitreous, interpreted as representing a state of synchysis. The lens was extracted with a loop. The eye quieted down after a lengthy period of inflammation. In the course of prolonged observation, the author became aware of a distinct difference of structure between the upper part of the vitreous through which the details of the retina could be clearly seen, and the subjacent part of the vitreous, which was cloudy and gave a bluish reflex. The plane of separation was almost horizontal, and the cloudy vitreous showed a wavy surface. It reached forward to the anterior chamber and backward to the posterior pole of the eye. The general effect was comparable to that produced by a vitreous hernia in the anterior chamber, or by the submerged portion of a fragment of ice in water. The vision of the eye remained poor, but was sufficient to enable the patient to find her way around her own home.

W. H. Crisp.

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Alagna, Gaspare. **The effect of prolonged administration of thyroid upon the eye.** Rassegna Ital. d'Ottal., 1940, v. 9, July-Aug., pp. 446-475.

Alagna fed hens large doses of thyroid in tablet form, and also fresh thyroid gland. Degenerative changes were found in the retina and optic nerve, according to the amount and duration of treatment. Alagna believes that these changes were due to direct action of the poison upon the cellular nervous tissue. Also noted was depigmentation of the uveal tract and sclera, explicable as a disturbance of the hormone action upon the sympathetic nervous system leading to an inhibition of pigment formation. (4 figures.) Eugene M. Blake.

Arndt, E. W. **Infection of sphenoidal sinuses with secondary infection of the eyes.** Brit. Jour. Ophth., 1941, v. 25, July, pp. 324-330. (See Section 10, Retina and vitreous.)

Bloom, W. A., Leech, M. P., and Shaw, W. J. **Temporary blindness due to sulfathiazole.** Jour. Missouri State Med. Assoc., 1941, v. 38, June, p. 202.

The authors report a case of temporary blindness in a white man, aged 26 years, who had been given for a kidney infection 1 gram of sulfathiazole every four hours for five days. Fourteen days after the drug was discontinued, the vision had returned to 20/20.

Theodore M. Shapira.

Korovitski, L. K., and Shein, I. S. **Therapy of ocular disease caused by plasmocide.** Viestnik Opht., 1940, v. 17, pt. 6, p. 747.

Satisfactory therapy, according to the authors, consists of pilocarpine used locally, caffeine-benzoate injections daily, and intravenous injections of hypertonic salt solution every other day.

Ray K. Daily.

Moorhouse, J. H. **A case of cyanopsia.** Trans. Ophth. Soc. of United Kingdom, 1940, v. 60, p. 230.

A young soldier was recovering from pneumonia and acute glomerular nephritis. In the fourth week the vision suddenly became hazy and a blue atmosphere surrounded everything. These attacks occurred four or five times, the longest lasting twenty minutes, during which time he was totally blind except for a very vivid blue. No drug had been given. The visual acuity of each eye was 6/6 a month later and no further attacks had occurred. The cyanopsia was believed to have been due to some uremic poison.

Beulah Cushman.

Riser, R. O. **Marble bones and optic atrophy.** Amer. Jour. Ophth., 1941, v. 24, Aug., pp. 874-878.

Vorisek, E. A. **Hereditary craniofacial dysostosis.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1014-1018.

Vorisek, E. A. **Ocular hypertelorism of Greig.** Amer. Jour. Ophth., 1941, v. 24, Aug., pp. 928-932.

13

EYEBALL AND ORBIT

Angius, Tulleo. **Spontaneous orbital hemorrhage.** Rassegna Ital. d'Ottal., 1940, v. 9, March-April, pp. 243-256.

A girl of 13 years presented an orbital hemorrhage at the time the third menstrual period should have occurred. No other cause could be found. Massive doses of ascorbic acid were followed by recovery. The author warns against exploratory puncture or other exploration of the orbit in such cases. A review of other causes of spontaneous orbital hemorrhage is given.

Eugene M. Blake.

Ayres, Francisco. **History of total transplantation of the eyeball.** Arquivos Brasileiros de Oft., 1940, v. 3, Dec., pp. 305-310.

A brief summary of the literary records of unsuccessful experiments. (20 references.)

Benedict, W. L. **Hyperostosis of the orbit.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1005-1012.

Geis, Franz. **Unilateral pulsating exophthalmos caused by rupture of the carotid artery in the cavernous sinus of the opposite side.** Klin. M. f. Augenh., 1941, v. 106, Feb., pp. 209-220.

A bullet entering the right temple caused pulsating exophthalmos of the left eye. If the carotid artery in the right cavernous sinus is injured, the venous blood cannot drain off through the superior ophthalmic vein of the same side. The arterial blood goes through the intercavernous sinus and the veins of the orbit of the opposite side, resulting in the pulsating exophthalmos. In the described case ligation of the right carotid artery considerably reduced the exophthalmos of the left eye. (5 illustrations.)

Gertrude S. Hausmann.

Kistner, F. B. **Decompression of exophthalmos.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, v. 25, pp. 118-123.

Kistner reviews the distressing and tragic sequence of events resulting from progressive exophthalmos caused by toxic goiter (corneal ulceration, stretching of the nerve, choked disc, atrophy, and even meningitis following ruptured corneal ulcers). He operated upon one case by the Naffziger method, by which the bone is removed from the floor of the anterior fossae, and upon two cases by the Sewell method, utilizing the space occupied by the sinuses to gain room for the orbital contents. (3 case reports.)

Lawrence G. Dunlap.

Kramelashvili, H. G. **Stump for prothesis, and optico-ciliary neurectomy.** Viestnik Opht., 1940, v. 17, pt. 6, p. 800.

The author believes that the postoperative course after ocular exenteration is better if an optico-ciliary neurectomy is done at the same time. He reaches the optic nerve by tenotomizing the externus. In cases where ordinary exenteration is not feasible, he enucleates the eyeball, exenterates it, replaces the scleral shell in Tenon's capsule, and covers it with conjunctiva.

Ray K. Daily.

Pasternak, H. G. **Tuberculous panophthalmitis.** Viestnik Opht., 1941, v. 18, pt. 1, p. 31.

A report of a case with detailed histologic data, the twelfth case so far reported in the literature. A 56-year-old man with disseminated tuberculosis developed subacute panophthalmitis of the left eye. In panophthalmitis of other etiology perforation of the eyeball leads to phthisis, while in tuberculous panophthalmitis the necrotic process continues after perforation. The latter was true in the present case.

Ray K. Daily.

Reichling, W., and Marx, H. **Dysthyrosis with severe ocular changes.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 374-407.

Five cases of exophthalmos in thyroid disease are reported. These show, according to the authors, no parallelism between the ocular and general symptoms. The first case, eleven years after the first thyrotoxic symptoms, developed exophthalmos which continued to progress despite lowering of the basal metabolism to normal under rest treatment. In the second case the exophthalmos was accentuated following thyroidectomy and the development of myxedema. The ocular symptoms were unrelieved by treatment with posterior pituitary hormone and diuretics, but improved under thyroxin and plastic operation on the lids. In the third patient the exophthalmos increased following thyroidectomy and the development of tetany. The general condition was improved under luminal and a protein-poor diet, but the exophthalmos was not influenced. The fourth patient had exophthalmos with thyrotoxicosis and metabolic disturbances of hypophyseal origin. The general condition but not the eyes improved with iodine treatment. In the fifth case the result was inconclusive.

In extreme exophthalmos with chemosis and strangulation of the conjunctiva, the authors recommend free excision of the conjunctiva in the four quadrants. They especially emphasize that this must be done before irreparable damage has been done to the tissue.

Frances C. Cogan.

Rumantzeva, A. F. **Prothesis for the orbit and adjacent structures.** Viestnik Opht., 1940, v. 17, pt. 6, p. 802.

The author describes the department for the manufacture of prothesis to

cover defects about the eyes and face, which was established at the Kiev Institute for Ophthalmology in 1938. (Illustrations.) Ray K. Daily.

14
EYELIDS AND LACRIMAL APPARATUS

Barkhash, S. A. **The placenta as plastic material in ocular surgery.** Viestnik Opht., 1940, v. 17, pt. 6, p. 758.

In 54 operations for trichiasis and pannus, placental tissue, preserved on ice, was used instead of mucous membrane from the lip. Satisfactory results were obtained. Ray K. Daily.

Burmistrova, E. **Surgery of cicatricial entropion.** Viestnik Opht., 1941, v. 18, pt. 1, p. 89.

A brief analysis of 407 operations for trachomatous entropion by Paradoksov's modification of the Panas operation. Ray K. Daily.

Decoud, A. C., and Schujman, S. **Palpebral sporotrichosis.** Anales Argentinos de Oft., 1940, v. 1, Oct.-Nov.-Dec., pp. 422-425.

The condition began with appearance of small papules which became pustular at the center. When the patient came for examination, the lower right lid showed a linear lesion extending horizontally the length of the lid, mostly of a dull violet color, with a finely scaly surface at the periphery, somewhat protruding at the center, and with hyperkeratosis. The diagnosis was confirmed by culture and by prompt improvement under the use of iodide of potash. The three illustrations show respectively epidermal hyperplasia, tuberculoid infiltration, and masses of plasma cells. W. H. Crisp.

Farina, F. **Hypophyseal adenoma and epiphora.** Rassegna Ital. d'Ottal., 1940, v. 9, Nov.-Dec., pp. 719-729.

A woman of 25 years complained of obstinate epiphora of eight months duration, for which no cause could be found and which resisted all local and general treatment. Eventually symptoms of pituitary dysfunction appeared and signs of acromegaly were evident. X-ray treatment resulted in marked general improvement and gradual disappearance of the epiphora.

Eugene M. Blake.

Kantor, D. B. **Late results of the author's modification of Sie-Boen. Lian's operation for trachomatous entropion.** Viestnik Opht., 1941, v. 18, pt. 3, p. 301.

A review of the surgery of trachomatous entropion and a report of the results of the author's technique. The author's modification consists in adding a vertical incision at each end of the longitudinal tarsotomy. The procedure achieves straightening of the tarsus, formation of an intermarginal space, and separation of the eyelashes from the eyeball. A follow-up of the cases shows that recurrence is frequent in the lower lid, because of the small size of the tarsus. For the same reason the operation is not suitable to upper lids with small tarsi. Ray K. Daily.

Kositzin, H. V. **Canaliculorhinostomy for epiphora following extirpation of the lacrimal sac.** Viestnik Opht., 1940, v. 17, pt. 6, p. 762.

This operation, described by Arruga in 1935 (Amer. Jour. Ophth., 1936, v. 19, p. 277), was performed on ten cases, with restoration of physiologic function in six. (Illustrations.)

Ray K. Daily.

Law, B. B., and Philip, J. F. **Aminoplastin as a conjunctival graft.** Brit. Med. Jour., 1941, April 5, p. 514.

Following an injury, a man aged 54 years developed a malignant melanoma of the palpebral conjunctiva of the left lower lid. The tumor was excised and the area treated with radon seeds. Suspicion of recurrence made it necessary to excise the lower lid, which was reconstructed in the following manner: A flap of aminoplastin was sutured to the lower edge of the cut bulbar conjunctiva and also to the skin at the lower edge of the wound, so as to form a deep fornix. The free end was then reflected upward and sutured to the upper border of a pedicle flap from the left temple, which was swung over to form the new lower lid. In this manner the inferior culdesac was formed by a double layer of aminoplastin. After three months there had been some shrinkage of the artificial conjunctiva at the outer canthus, forming a symblepharon, which, however, had given no discomfort. John C. Long.

Morgan, A. L. **Plastic repair of deformities of the eyelids.** Canadian Med. Assoc. Jour., 1941, v. 44, June, p. 560.

The repair of three typical cases of lid deformity is described. The first case was of cicatricial ectropion. The scar was incised, allowing the skin edges to gape rather widely. The skin defect was filled with a free full-thickness graft of skin obtained from the opposite upper eyelid. Temporary adhesions between the lids were produced to prevent retraction. The second case was one of congenital ptosis with epicanthus. The epicanthal fold was cut through horizontally and sutured vertically. The ptosis was then corrected with a fascia-lata implant according to the method of Wright. The third case was one of marked congenital coloboma of the upper lid. This was corrected by a sliding full-thickness flap from the temple after first forming an upper fornix from bulbar conjunctiva. The author suggests that plastic surgery around the eye should be considered the province of the ophthalmologist.

John C. Long.

Regoli, Attilio. **The effect of testicular extract (factor R) upon anesthesia in cicatricial tissue of the eyelid.** Rassegna Ital. d'Ottal., 1940, v. 9, July-Aug., pp. 433-445.

Regoli reports upon the effect of testicular extract dropped into the conjunctival sac and novocaine injected into the tissues in three cases of cicatricial ectropion. He demonstrates that anesthesia is more profound and persists longer by this method. Animal experimentation is discussed, and a review of the literature is added.

Eugene M. Blake.

Resnik, Z. I. and Kozshuknova, A. P. **Roentgen therapy of phlegmon of the lacrimal sac.** Viestnik Opht., 1940, v. 17, pt. 6, p. 804.

The merits of this conservative form of treatment are its harmlessness, painlessness, and the cosmetic end result. Of 41 cases treated with X rays, 21 recovered, 11 improved, and in 8 the treatment had no effect.

Ray K. Daily.

Savin, L. H. **Tarsectomy for trachoma, its indications, difficulties, and results.** Trans. Ophth. Soc. United Kingdom, 1940, v. 60, p. 163. (See Section 5, Conjunctiva.)

15

TUMORS

Costa Aguiar, Paulo da. **Neoplasm of the caruncle.** Arquivos Brasileiros de Oft., 1941, v. 4, June, pp. 140-142.

The patient was a boy of twelve years. The tumor had been first noticed two months previously, and had grown

rapidly. The patient came on account of acute hemorrhage from the growth and simultaneously from the corresponding nostril. At this time the growth was found to be double, both parts being pedunculated, with the "body" protruding outside the lids. The laboratory diagnosis was of a "benign histiocytic tumor, probably xanthomatous in origin"; but the author regrets that inclusion of the tumor in paraffin prevented elective staining for confirmation or otherwise of this diagnosis.

W. H. Crisp.

Cristini, Giuseppe, **Multiple pigmented epithelioma of the conjunctiva.** Rassegna Ital. d'Ottal., 1940, v. 9, Nov.-Dec., pp. 688-701.

A multiple pigmented growth involving the right eyelid and contiguous bulbar conjunctiva of a 43-year-old woman is reported. Because of the histology, and the genesis of the pigment, the author holds that this blastoma must be classified with pavement epitheliomata. The pigmentation is believed to be a phenomenon of the second order and due to the presence of Langerhans cells. (7 figures.)

Eugene M. Blake.

Elder, N. G. **A neurofibroma of the oculomotor nerve.** Jour. Path. and Bact., 1941, v. 52, March, pp. 263-264.

A solitary neurofibroma of the oculomotor nerve was found in an anatomic specimen, the clinical and postmortem findings being unknown. The tumor was small and circumscribed, and was located in the intracranial portion of the nerve. The histologic appearance was typical of neurofibroma.

T. E. Sanders.

Gilbert, W. **The biology of malignant melanoma of the choroid.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 549-553.

Melanosarcoma of the choroid should not be classified according to Knapp's four stages, since metastasis may occur in any stage. The author emphasizes the fact that whether or not a melanosarcoma metastasizes depends on the biologic reaction of the general organism to it. He therefore suggests classifying melanosarcoma of the choroid into: (1) the premetastatic, and (2) the metastatic stage.

Frances C. Cogan.

Giqueaux, R. E. **Epithelioma of the sclerocorneal limbus.** Anales Argentinos de Oft., 1941, v. 2, March, pp. 1-5.

In a man of 66 years, the tumor, after three-years development, extended from the center of the cornea to the caruncle of the left eye. It was whitish in color, and had an irregular, globulated surface. Biopsy established the diagnosis of epithelioma. (2 illustrations.)

W. H. Crisp.

Goedbloed, J., and Wÿers, H. J. G. **Follicular lymphoblastoma.** Acta Ophth., 1941, v. 19, pt. 1, p. 28.

A man 68 years old developed symmetrical orbital tumors. The general examination was negative. Thirteen years previously he had had a swelling in the groin and enlargement of the pelvic glands, both of which disappeared under X-ray therapy. Excision of the tumors was followed by recurrence with retrobulbar extension and exophthalmos. The exophthalmos receded on X-ray irradiation. (Illustrations.)

Ray K. Daily.

Lefkoeva, E. F. **A review of the histogenesis of so-called sarcoma of the uveal tract.** Viestnik Opht., 1940, v. 17, pt. 6, p. 725.

A detailed analysis of the pathologic material (202 uveal tumors) of the Helmholtz Institute. The author concludes that the structure of the tumor, the character of the cellular elements, the type of growth, and the age of the patient relate this neoplasm to tumors of the nervous system. She suggests therefore that the neoplasm be designated as a malignant melanoma or meningioma. (Photomicrographs.)

Ray K. Daily.

Martin, J. P., and Savin, L. H. **A case of tuberous sclerosis with "phacomata."** Brit. Jour. Ophth., 1941, v. 25, July, pp. 305-313.

The authors review the literature on the subject in point, and report a case contributing to the general interest. A man aged 34 years had been epileptic and mentally defective since childhood. There was an extensive adenoma sebaceum of the face. He had made no complaint of blindness, but had been sent to the hospital from an epileptic institution because of his walking into doors, tables, and similar objects. Ophthalmic examination showed ocular movements to be normal. Light perception was present. Further details as to vision were obscure by reason of the mental state, but it was the opinion of the ward sister that the man had little useful vision. The discs showed grayish pallor, blurred margins, and swelling. On the upper nasal aspect of the left fundus was a small, glistening tumor with a mulberry surface. In the retina along the nasal aspect was a second, white swelling, elongated in shape. The cerebrospinal fluid showed a reading lower than normal but with greatly increased protein content. Clinical findings convinced the authors that the case was one of tuberous sclerosis and that the globular tumor on the left disc and the large left retinal tumor were phacomata. It was also their opinion that small white swellings found in the right fundus were phacomata. Loss of vision is believed to have been associated less with the phacomata than with the changes in the discs themselves. (Illustrations, references.) D. F. Harbridge.

Pacheco Luna, R. **A case of bilateral metastatic carcinoma of the choroid.** Guatemala Med., 1941, v. 6, March, pp. 2-4.

The patient, a woman of 46 years, had had hysterectomy for cancer of the womb five years previously, and removal of a carcinoma of the breast three years after the first operation. More recently there had been recurrence locally in the breast, and diagnosis of metastases in the lungs and brain. Each eye showed a tumor at the posterior pole, the eyes being entirely blind. W. H. Crisp.

Pokrovskii, A. I. **Angioma of the orbit, and its surgery.** Viestnik Opht., 1941, v. 18, pt. 3, p. 235.

A report of four cases, and a discussion of the surgical approaches to the orbit. These cases illustrate the functional disturbances produced by such neoplasms, and the favorable effect of their removal. The earlier extirpation can be done, the easier it is to perform, and the greater the practicability of doing it through a simple orbitotomy. (Illustrations.) Ray K. Daily.

Pokrovskii, A. I. **Neoplasms of the orbit.** Viestnik Opht., 1941, v. 18, pt. 2, p. 115.

A report of four cases of chloroma of the orbit. Two were fatal, and the diagnosis was confirmed histologically. Two disappeared from observation.

The patients were 8, 4, 7, and 15 years of age, respectively. (Illustrations.)

Ray K. Daily.

Pokrovskii, A. I. **Neoplasms of the orbit.** Viestnik Opht., 1941, v. 18, pt. 1, p. 3.

A review of the literature and a report of a fatal case of lymphosarcoma, which infiltrated the retrobulbar space and adjacent tissues. When first seen the tumor was so extensive that the case was considered hopeless. (Illustration.) Ray K. Daily.

Rumbaur, W. **Retinal angiomatosis.** Klin. M. f. Augenh., 1941, v. 106, Feb., pp. 168-198.

Five cases in one family represented different stages of this condition. Two of the cases showed only small angiomas, another spontaneous regression, and still another regression in one eye while the other eye became blind through detachment of the retina. In the fifth case one eye had been enucleated following a detachment. An attempt was made to stop the growth of the angioma and the progress of the detachment by diathermic coagulation, but the case was too advanced. (5 illustrations, 1 genealogical tree.)

Gertrude S. Hausmann.

Sheplakova, V. M. **Dictioma of the retina.** Viestnik Opht., 1941, v. 18, pt. 3, p. 323.

A report of a case of this neoplasm, which arises from the unpigmented ciliary epithelium. An infant nine months old was brought with buphthalmos of the left eye. The eye was enucleated. Within a year there was recurrence in the orbit and regional glands. The infant died one day after the orbit was exenterated. The microscopic picture was identical in the intraocular neoplasm and in the orbital recurrence. The infiltrating character, the number of mitoses, the foci of necrosis, and the rapid growth indicate its malignant character. Ray K. Daily.

Siotto, Giovanni. **Reticuloma of the conjunctiva.** Rassegna Ital. d'Ottal., 1941, v. 9, July-Aug., pp. 477-492.

Siotto discusses the development of our knowledge of tumors of the reticulo-histiocyte system and reviews the literature to date. He reports a tumor involving the right upper lid of a 17-year-old girl. The microscopic characteristics are minutely described (9 figures.) Eugene M. Blake.

Smelanski, P. I. **Retinal angiomatosis.** Viestnik Opht., 1940, v. 17, pt. 6, p. 740.

Smelanski reports five cases, in two of which the disease was confined to the retina. One case was under observation from the time of the inception of the disease until the visual loss was total, a period of five years. This patient had accessory nipples and hypospadia, indicating a widespread interference with normal development. Anatomic study of the five cases demonstrated that the primary process was vascular. The secondary changes (exudates, cysts, and retinal detachment) appeared much later. In the majority of the cases the first changes were seen in the upper-outer retinal quadrant, and in only one case did the disease begin in the central portion of the retina. In one case the involvement was far in the periphery and could easily have escaped detection without a very thorough ophthalmoscopic examination. This suggests that many cases may escape diagnosis. Ray K. Daily.

Strachov, V. P. **Recklinghausen's disease, neurinoma of the right orbit, and neoplasm of the left choroid.** Viestnik Opht., 1941, v. 18, pt. 1, p. 12.

A man 35 years of age was found to present this combination of pathologic processes. The author believes that the case throws some light on the histogenesis of uveal sarcoma. The presence of three pathologic processes in the same patient may be attributed to disturbance in the primordial layer. Since Recklinghausen's disease and neurinoma are known to be ectodermal in origin, it appears probable that sarcoma of the choroid is caused by ectodermal elements which have migrated into the mesodermal layer. (Photomicrographs.) Ray K. Daily.

Tiscornia, B. J., and Lami, I. **Epithelioma of the conjunctiva; radium therapy.** La Semana Med., 1941, v. 48, Feb. 27, pp. 504-511.

The growth, in a woman of 63 years, had existed for three years and appeared as an infiltrating ulcerated mass between the sclerocorneal limbus and the outer canthus, with adhesions between the eyeball and the internal surface of each eyelid. The cornea was deeply infiltrated, and vision was reduced to seeing shadows at 2 meters. After biopsy, which was preceded by roentgen therapy, radium tubes were applied several times in the course of nine days, to a total of 93.60 mcd. of radium element. Complete atrophy of the tumor resulted. W. H. Crisp.

16
INJURIES

Astakhova, A. P. **Ophthalmoscopic investigations on dogs in experimental fatal anemization and resuscitation.** Viestnik Opht., 1941, v. 18, pt. 2, p. 149.

Experimental anemia to the point of cessation of cardiac and respiratory activity was produced by venesection. Clinical death was timed at the moment of the last agonal breath. Five to ten minutes after the clinical death resuscitation was begun by transfusion of the collected blood and artificial respiration. The tabulated ophthalmoscopic data relative to the fundus, tension, pupillary size, corneal transparency and sensitivity, show that the eye is very sensitive to anemia. The first sign is a disturbance in retinal circulation, which appears early, when the animal loses about one half its blood volume. The ophthalmoscopic picture and tension during the interval between clinical death and beginning of resuscitation correspond to those in the eye of a dead animal. The pupillary and corneal reflexes disappear long before clinical death, and reappear twenty minutes after the beginning or resuscitation. Edema of the deep tissues could not be demonstrated, but all cases had a light corneal dullness suggestive of endothelial edema. Ray K. Daily.

Bakker, A. **A transient but noteworthy traumatic complication of local contusion of the eye.** Ophthalmologica, 1941, v. 101, June, p. 364.

In an eye that had been struck with a small steel spring, a punctate hemorrhage resulted in the conjunctiva just above the limbus at the 12-o'clock position. For a half-hour the pupil was narrowly pyriform with its apex at vertical. One does not like to assume that contusion can produce both paralysis of the sphincter and stimulation of the dilator fibers. The author's observation, however, makes it reasonable to accept sympathetic stimulation as a cause of

traumatic mydriasis when sphincter paralysis does not seem a likely explanation. F. Herbert Haessler.

Baltin, M. M., and Sviadocz, B. I. **X-ray diagnosis in blunt and sharp firearm wounds of the orbit.** Viestnik Opht., 1940, v. 17, pt. 6, p. 786; and 1941, v. 18, pt. 3, p. 306.

This very detailed and exhaustive study, illustrated with case reports, leads to the following conclusions: X-ray examination in firearm injuries to the orbit has diagnostic, prognostic, and therapeutic significance. In forty-eight cases of orbital emphysema the air was located chiefly in the upper lid, and was due to injury to the ethmoids, a fracture of the lamina papyracea permitting the entrance of air from the ethmoids into the orbit. The X-ray plates show that Heerfordt was wrong in believing that the air was retrobulbar. In all cases of traumatic enophthalmos fracture of one of the orbital walls was demonstrated, the inner orbital wall being most frequently injured. Tears in the orbital fascia are probably also significant in producing enophthalmos. In cases of pulsating exophthalmos special attention should be directed to the superior orbital fissure and the sella turcica. Widening of the superior orbital fissure indicates dilatation of the ophthalmic veins, and changes in the sella turcica indicate dilatation of the cavernous sinus. X rays afford a differentiation between true and spurious pulsating exophthalmos, and permit observation of the dynamics of the process. Roentgenography can demonstrate fractures through the optic canal, and is invaluable in locating foreign bodies in the orbit, in the cranium, and in the sinuses. (Illustrations.)

Ray K. Daily.

Baquis, Mario. **A peculiar fundus lesion following contusion of the globe.** Rassegna Ital. d'Ottal., 1940, v. 9, May-June, pp. 352-362.

In a 16-year-old boy, a blow on the left eye by a tennis ball produced an irregular pupil and an oval, brownish, edematous area, resembling commotio retinae, in the macula. After two weeks the retinal lesion changed to a sharply defined, irregular, white area. At the end of two months, this had cleared up entirely. The author discusses the pathology of fundus changes resulting from contusion by blunt objects. (2 figures.) Eugene M. Blake.

Berrettini, G. L. **Mydriatic and cycloplegic principle existing in "trombeteira" (Datura arborea and D. fastuosa L.).** Arquivos Brasileiros de Oft., 1941, v. 4, Feb., pp. 50-59. (See Section 2, Therapeutics and operations.)

Cauer, R. **Moving piece of glass for 23 years in the eyeball.** Klin. M. f. Augenh., 1941, v. 106, Jan., pp. 91-93.

Twenty-three years after a perforating injury 9 mm. behind the limbus, the patient's eye became inflamed and a piece of glass about 4 mm. long was found lying on the iris. The glass was extracted and the eye healed normally.

Gertrude S. Hausmann.

Chepurina, T. S. **Electric cataract.** Viestnik Opht., 1941, v. 18, pt. 2, p. 202.

A report of a case of electric cataract which developed 2½ months after a severe electric shock in a man 21 years old. The patient's right eye was burned and had to be enucleated. The left lens became rapidly opaque and was removed uneventfully by linear extraction. Ray K. Daily.

Colditz, H. **Contribution to the better visibility of foreign bodies in the posterior wall of the eyeball and their operative removal with the use of the scleral lamp.** Klin. M. f. Augenh., 1941, v. 106, Jan., pp. 94-99.

A new method is discussed in which air is insufflated into Tenon's space and simultaneously the Comberg prothesis is used. Diascleral transillumination is very valuable in localizing the foreign body. Gertrude S. Hausmann.

Dashevskii, A. I. **Scleral projection of intraocular foreign bodies.** Viestnik Opht., 1941, v. 18, pt. 1, p. 83.

A detailed description of the mathematical projection of an intraocular foreign body upon the sclera, from roentgenographic data. The roentgenographic projector, designed by the author, eliminates this mathematical task and acts on the principle of a slide rule. (Illustration.) Ray K. Daily.

Fieandt, Olof. **A case of transitory myopia, following radium irradiation.** Acta Ophth., 1941, v. 19, pt. 1, p. 11.

Following radium irradiation of a flat epithelioma of the limbus, the twenty-year-old patient developed a transitory myopia of eight days duration. A search of the literature failed to find a similar case. Ray K. Daily.

Geilikman, O. B. **The late extraction of intraocular foreign bodies.** Viestnik Opht., 1941, v. 18, pt. 2, p. 183.

A review of the material of Helmholtz Institute for 1939, which consisted of 49 cases in which extraction was delayed because the patients lived in localities distant from institutions equipped for this type of surgery. In 41 cases the foreign body was extracted, and in eight the operation failed. A review of the clinical histories leads to the following conclusions: small iron splinters should be extracted in all cases, because their prolonged intraocular retention leads to siderosis and loss of vision; the extraction of small magnetic splinters is possible only transclerally, which requires accurate localization. Foreign bodies in the ciliary body should be extracted by an incision over the ciliary body. Foreign bodies retained in the eye for long periods can be extracted, with the availability of accurate localization and the exercise of surgical perseverance.

Ray K. Daily.

Horner, W. D. **The handling of non-industrial eye injuries with illustrative cases.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, v. 25, pp. 96-106.

The author discusses a wide variety of primary and secondary injuries to the eye including those due to traffic and household accidents, alcoholism, fights, and robberies. Hemostasis, early treatment to prevent infection, reposition, nutrition, and rest are stressed.

Lawrence G. Dunlap.

Kahn-Ostwald, and Joseph, E. **Unilateral edematous and exudative neuroretinitis appearing after removal of a corneal foreign body.** Bull. Soc. d'Opht. de Paris, 1939, June, p. 338.

The day following the removal of a tiny superficial foreign body from the cornea, a 35-year-old man complained of sudden failure of vision. An extensive neuroretinitis was found on examination. Exhaustive general and laboratory investigation failed to establish any definite cause for the condition. The authors speculate on the possible relation of the foreign body to the intraocular process.

George A. Filmer.

Kolen, A. A. **The scleral incision of transcleral magnet extractions.** Viestnik Opht., 1941, v. 18, pt. 1, p. 76.

The author dissects a pocket between the sclera and choroid, believing that the introduction of the magnet into this pocket facilitates unimpeded attraction of the foreign body. Ray K. Daily.

Larsson, Sven. **Bloody bone-free roentgenography of the eyeballs.** Acta Ophth., 1941, v. 19, pt. 1, p. 1.

The author points out the advantages of this method, which was described by Franceschetti in 1934. The application of the X-ray film directly to the exposed sclera affords such precise localization that it is at times possible to remove even non-magnetic foreign bodies, by cutting down directly over them. Two such cases are reported.

Ray K. Daily.

Machlin, I. M. **The effect of sand, alabaster, and cement on the eye.** Viestnik Opht., 1941, v. 18, pt. 2, p. 193.

Laboratory investigation on rabbits showed that sand and alabaster form no chemical combination with ocular tissues and act as foreign bodies. Cement through its lime content burns the eye, but less severely than lime. Immediate profuse irrigation with cold water is adequate for first-aid.

Ray K. Daily.

Medvedev, H. I. **Combined method of roentgenographic localization of intraocular foreign bodies.** Viestnik Opht., 1941, v. 18, pt. 2, p. 134.

A detailed description of the mathematical calculation based on data derived from antero-posterior and sagittal X-ray plates of the eyeball, with the center of the cornea marked by a drop of bismuth. (Illustrations.)

Ray K. Daily.

Pergola, Alfredo. **Traumatic scleral rupture with aniridia and subconjunctival luxation of the lens.** Rassegna Ital., d'Ottal., 1940, v. 9, Nov.-Dec., pp. 626-644.

The author cites the various forms of scleral rupture reported in the literature and describes one personally observed case. He reviews the various theories which attempt to explain the mechanism of indirect traumatic rupture, and offers an explanation of the process resulting in aniridia and subconjunctival luxation of the lens.

Eugene M. Blake.

Plitas, P. S. **A simplified method of roentgenographic localization of intraocular foreign bodies.** Viestnik Opht., 1941, v. 18, pt. 1, p. 79.

To facilitate localization, the author uses a lead marker sutured to the conjunctiva and injections of radiopaque substances into Tenon's capsule.

Ray K. Daily.

Spivakovskii, D. H. **Results of juvenile ocular traumatism.** Viestnik Opht., 1940, v. 16, pt. 6, p. 807.

The author reviews the material (184 severe injuries) of the Odessa Eye Clinic for the years 1933 to 1937. The conclusions are that such injuries are usually the result of carelessness and unsupervised play. The data show that with better supervision of children's recreation the number of injuries is diminishing from year to year. The number of injuries is greatest in children from 9 to 12 years old, and is three times greater in boys than in girls. Of the injuries, 41.8 percent were made with blunt objects, 27.7 percent with cutting objects, 15.9 percent with firearms and explosives, and 15.9 percent were chemical and heat burns. The visual results were better in cases in

which ophthalmologic service was promptly available. Ray K. Daily.

Talbot, Parlange, and Duroux. **A case of traumatic enophthalmos.** Bull. Soc. d'Opht. de Paris, 1939, Feb., p. 72.

Four months after a blunt trauma to the eye and periorbital region, a patient developed enophthalmos, conjunctival injection, photophobia, miosis, and paresis of the external rectus. Because some of the factors in the clinical picture were those seen in Horner's syndrome, the authors presume a sympathetic origin of the condition.

George A. Filmer.

17

SYSTEMIC DISEASES AND PARASITES

Aires, Francisco. **Ocular disturbances in diabetes.** Arquivos Brasileiros de Oft., 1941, v. 4, June, pp. 127-132.

A brief communication with two case reports.

Ajo, C. **A new observation on a primary ocular reaction to Shwartzman toxins.** Proc. Soc. Exper. Biol. and Med., 1941, v. 47, June, pp. 400-401.

The author reports a marked reaction in the eye of the rabbit following intravenous injection of Shwartzman toxins, which are culture-filtrates of meningococcus and B. typhosus. The reaction consists of miosis, photophobia, lacrimation, and congestion of the iris and conjunctiva, with a marked pericorneal ring of dilated capillaries and in some instances with gross subconjunctival hemorrhages. Microscopically, severe reactions show massive conjunctival hemorrhage with dilatation and engorgement of blood vessels of the recti muscles. No lesions have been seen in the iris, retina, cornea, or ciliary body. The active factors in the filtrates may be inactivated by immune neutralizing serum. T. E. Sanders.

Ballantyne, A. J. **Ocular complications in hyperemesis gravidarum.** Jour. Obstet. and Gynec. British Empire, 1941, v. 48, April, pp. 206-219.

A short survey is given of the incidence, etiology, pathology, and treatment of hyperemesis gravidarum. Ballantyne reports a series of six cases with ocular complications of varying degrees of severity ranging from slight dimness of vision, without ophthalmoscopic changes, to total blindness, with gross hemorrhages and optic neuritis. He believes that the earliest ocular disturbance is a retrobulbar neuritis associated with a central field defect, without visible ophthalmoscopic changes. This is succeeded by a visible edema of the optic nerve and later by a characteristic type of peripapillary retinal hemorrhage. In the treatment of the condition the author recommends the routine method of rest and restoration of fluid loss, supplemented by intramuscular administration of vitamins B_1 and C. If this procedure does not give a rapid response, especially if a polyneuritis and the above eye signs are present, prompt termination of pregnancy is indicated.

T. E. Sanders.

Cristini, Giuseppe. **Ocular changes in acute leukemia.** Rassegna Ital. d'Ottal., 1940, v. 9, Sept.-Oct., pp. 578-605.

Cristini discusses the various conceptions of leukemia and quotes the views of outstanding investigators. He presents the history and microscopic pathology for each of four cases. Clinically, the outstanding feature was retinal hemorrhage, while pathologically the majority of changes affected the choroid as a leucocytic infiltration of

the stroma, ascribed to venous capillary stasis. The outstanding feature of a hyperplastic nature was metaplasia of the cells of the vessel sheaths, which appeared as mesodermic cells of the uvea. There was also hyperplasia of the endothelial cells of the lymphatic spaces of the corneal trabeculae with an increase in the glial cells of the retina and optic nerve. (11 figures.)

Eugene M. Blake.

Dias, A. B. **Hypovitaminosis, its repercussion on vision.** Arquivos Brasileiros de Oft., 1941, v. 4, Feb., pp. 15-26.

A discussion of the sources and applications of vitamins, with special regard to ocular disorders as mentioned in the literature. W. H. Crisp.

Levin, E., and Hanono, M. **Ocular compression in paroxysmal tachycardia.** La Semana Med., 1941, v. 48, July 10, pp. 83-90.

Six cases of paroxysmal tachycardia are described and illustrated (with the inclusion of electrocardiograms), to demonstrate the therapeutic value of strong compression of the eyeball in relieving the attacks. W. H. Crisp.

Marchesani, O. **Rheumatic inflammations of the eye.** Klin. M. f. Augenh., 1941, v. 106, Jan., pp. 1-20.

Rheumatic origin is considered in inflammations of the sclera, cornea, and iris. These conditions are believed to be an allergic reaction in the tissues of the body, which may be caused by tubercle bacilli, gonococci, staphylococci or, most frequently, streptococci. Depending on the immune biologic condition at the time, the infection may be an isolated disease of one organ or a generalized infection. (8 illustrations.) Gertrude S. Hausmann.

Meyer, F. W. **The histology of human and experimental tuberculosis with special emphasis on tuberculous retinitis.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 408-499.

Clinical and histologic studies of 33 tuberculous human eyes and of experimental ocular tuberculosis in rabbits form the basis of this report. The animals were first sensitized by a generalized tuberculous infection and then inoculated with an avirulent human strain in the ciliary vein. The resultant lesions in the animal eyes were essentially the same as in clinical tuberculosis.

Tuberculous iridocyclitis showed nonspecific infiltration adjacent to typical granulation tissue and caseation. Certain diagnosis was impossible in long-standing cases. In severe cases the sclera, choroid, and retina were usually involved with the iris and ciliary body. Retinal periphlebitis was in most cases found to accompany iridocyclitis. The perivascular infiltrate was seen microscopically to consist of round cells next to the vessels; these were surrounded by epithelioid and giant cells. The thickened vessel walls caused narrowing and sometimes obliteration of the lumina. Secondary changes such as hemorrhage and development of new blood vessels occurred in long-standing cases. There was also a marked epithelial hyperplasia which the author said might be confused with tubercle of the retina. The author found no evidence to support Marchesani's theory that retinal periphlebitis is due to thrombo-angiitis obliterans.

Frances C. Cogan.

Yousefova, F. I. **Metastatic ocular tuberculosis.** Viestnik Opht., 1940, v. 17, pt. 6, p. 714.

An analysis of 100 cases shows the multiform character of ocular tubercu-

lous lesions. In most of the cases there was stimultaneous involvement of several ocular structures, with a predominance of uveal disease. The focal reaction was indicative of involvement of the ciliary body, even in cases where the disease was clinically limited to the other layers of the eyeball. The author believes that the ciliary body plays an important role in the pathogenesis of the disease. In 86 percent of the cases there was some improvement, and with the regression of the inflammatory symptoms there was a significant rise in vision. Results were better in cases in which treatment was instituted early. In some cases in which therapy was ineffective and difficult the disease assumed a recurrent character. In hypersensitive cases desensitizing therapy, such as calcium, physiotherapy, and general tonic measures, was of great value in preparing the patient for the safe administration of tuberculin. (Illustrations.) Ray K. Daily.

18

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Berrettini, G. L. **Incidence of trachoma in S. José dos Campos (State of São Paulo).** Arquivos Brasileiros de Oft., 1940, v. 3, Dec., pp. 301-304.

A percentage of 6.51 among 399 patients in a private clinic of S. José dos Campos contradicts the general idea that the eastern zone of the State of São Paulo is free from trachoma. All the 26 trachoma patients were of the white race. W. H. Crisp.

Guglianetti, Luigi. **Occupational diseases of the eye.** Rassegna Ital. d'Ottal., 1941, v. 10, Jan.-Feb., pp. 3-115.

A very extensive review of the known facts regarding occupational diseases of the eye and not adaptable to abstract.

James, R. R. **Robert Brudenell Carter.** Brit. Jour. Ophth., 1941, v. 25, July, pp. 330-339.

A presentation of the scientific accomplishments of Robert Brudenell Carter, an intermediate between the giant founders of modern ophthalmology, such as Bowman and the elder Critchett, and the more modern consolidators of the science, such as Nettleship and William Lang. His birth was in 1828, his death in 1918. His works are reviewed, instances being cited to support his ability in surgery and his ease with the pen. (Photographs, references.)

D. F. Harbridge.

Kaminski, L. S. **Ocular diseases in Moscow.** Viestnik Opht., 1941, v. 18, pt. 3, p. 312.

A statistical analysis of the material of the eye clinics and hospitals for 1937. Ray K. Daily.

Kraehenbuehl, J. O. **Functional lighting in the college.** Sight-Saving Review, 1941, v. 11, March, p. 13.

To determine a satisfactory type of lighting and amount of illumination will require the coöperation of both the medical and engineering professions. The architect also must be consulted in carrying out the lighting plans. The author discusses in some detail the lighting requirements for class rooms, pointing out the more common desirable and undesirable situations encountered. Fluorescent lighting is discussed. Apparently this new type of lighting is not overwhelmingly superior to that supplied by filament globes, and a number of factors should be considered in deciding which type of illumination to

employ. The fluorescent system gives more light per watt than the filament system, but this saving is somewhat offset by the increased installation and maintenance cost. John C. Long.

Machlin, I. M. **Ninety years of the Helmholtz ophthalmoscope.** Viestnik Ophth., 1940, v. 17, pt. 6, p. 805.

A brief review of the development of the ophthalmoscope from the original invention to its present forms.

Ray K. Daily.

Meyer, I. C. **Heredity in ophthalmology.** Ophth. Ibero-Amer., 1940, v. 2, no. 1, pp. 7-21.

A 15-page survey of the subject, with special mention of the views of Treacher Collins in ophthalmology, and of the research of Morgan in the science of heredity as illustrated by the fruit fly. W. H. Crisp.

Nicholls, J. V. V. **A survey of ophthalmic conditions among rural school children.** Canadian Med. Assoc. Jour., 1941, v. 44, May, p. 472.

In October, 1939, an ophthalmic survey was carried out in Pontiac County, Quebec (Amer. Jour. Ophth., 1940, v. 23, p. 1306), and in May, 1940, a similar survey was conducted in this county and also in Wakefield County. In the first survey 29.7 percent of the children had defects of vision, whereas in the second survey only 14.6 percent were defective. The incidence of hypermetropia, hypermetropic astigmatism, and anisometropia was practically identical in the two surveys. The cause of the difference in the incidence of subnormal vision was entirely due to the smaller number of cases of strabismus and myopia found in the latter survey. The hereditary factor of both of these conditions is well recognized. In the first series more of the pupils were related than in the second survey. The second group of children examined were younger and hence had not developed myopia to the same degree as found in the older children; also, a high percentage of the children in this group attended rural schools which closed during the severe winter months, thereby decreasing the amount of intensive eye work done. John C. Long.

Pergola, Alfredo. **Trachoma among the inhabitants of Asmara.** Rassegna Ital. d'Ottal., 1940, v. 9, May-June, pp. 337-351.

The author discusses the cause of the diffusion of trachoma in Asmara and gives the relative percentage of the ocular involvements. He stresses the high percentage of trachoma among the schoolchildren.

Eugene M. Blake.

Prado, Durval. **The school as educational element in the struggle for the prevention of blindness.** Arquivos Brasileiros de Oft., 1941, v. 4, June, pp. 146-151.

The subject is concisely discussed under the following headings: factors in prevention of blindness related to heredity and prenatal hygiene; prophylaxis of trachoma; illumination; examination of refraction; myopia; prophylaxis against blindness in industrial accidents; classes for amblyopes.

W. H. Crisp.

Rocha, Hilton. **Ophthalmology within medicine.** Arquivos Brasileiros de Oft., 1941, v. 4, June, pp. 119-125.

Inaugural address of the medical course of the Paulist School of Medicine (Brazil).

Rodigina, A. M. **Ocular diseases and the organization of ophthalmologic service in some districts of Ydmurt.** Viestnik Opht., 1940, v. 17, pt. 6, p. 815.

A report of a survey of this trachoma-infested district. The decision is that the service is inadequate for the needs. Ray K. Daily.

Tzikulenko, K. I. **Classification of trachoma.** Viestnik Opht., 1941, v. 18, pt. 1, p. 36.

A review of the literature. The author considers classification on the basis of the conjunctival process inaccurate, because trachoma involves also other ocular structures. Epidemiologically patients with trachoma in the fourth stage should be provisionally considered cured and should be retained under observation. Ray K. Daily.

Weskamp, Carlos. **Ocular traumatology.** Anales Argentinos de Oft., 1940, v. 1, Oct.-Nov.-Dec., pp. 397-404.

A very general consideration of the subject, with the practical recommendation that the law ought to penalize simulation when proved.

W. H. Crisp.

19

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Brown, G. L., and Harvey, A. M. **Neuromuscular transmission in the extrinsic muscles of the eye.** Jour. of Physiology, 1941, v. 99, March 25, p. 379.

The responses to nerve stimulation of the extrinsic ocular muscles in decerebrated cats are reported in detail, the small size and uniform structure of these muscles making them particularly useful for electric recording. The refractory period, recovery, and facilitation phenomenon in normal muscles and in those treated with curarine, eserine, and acetylcholine were studied.

T. E. Sanders

Cibis, Paul. **Structure and histochemistry of the neuroepithelium of the human retina.** Klin. M. f. Augenh., 1941, v. 106, Feb., pp. 160-167.

The author was able to demonstrate in a histologic section of an eyeball three different shaded parts which formed the cones in the retina. With his method the differentiation between rods and cones was fairly easy to make. (One illustration.)

Gertrude S. Hausmann.

Lewis, J. T. **Sympathetic and parasympathetic innervation of the eye.** Anales Argentinos de Oft., 1940, v. 1, Oct.-Nov.-Dec., pp. 405-421.

A 17-page summary of the subject, with a bibliography of 37 references.

Martìnez Hinojosa, Francisco. **Pupillodilator centers.** Anales de la Soc. Mexicana de Oft. y Oto-Rino-Laring., 1940, v. 15, March-April, pp. 76-100.

This address, dedicated to the memory of Daniel Vèlez, devotes 25 pages to a survey of theories and anatomical knowledge of the subject. (10 references, 2 illustrations.) W. H. Crisp.

Reichling, W., and Klemens, F. **A vascular connective-tissue layer between the pigment epithelium of the retina and the lamina vitrea. Part 2.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 500-512.

In histologic sections of eyes from persons of fifty years or older, the authors found a layer of vascular connective tissue situated behind the ora serrata and the pigment epithelium.

The vessels in this layer connect directly with vessels of the choroid and the interlamellar connective tissue. The connection with the choroidal vessels, which lie on the outer layer of the lamina vitrea, occurs behind the ora serrata through holes in the lamina vitrea. The vascular connection with the interlamellar connective tissue is made by a direct extension of the vessels from this new tissue. The presence of vessels which enter into the interlamellar connective tissue from the new layer supports Wolfrum's theory that the interlamellar connective tissue is a continuation of the subpigment epithelium foundation substance.

Frances C. Cogan.

Talkovskii, S. I. **Comments on Sokolov's dissertation, "The ciliary nerves in the human."** Viestnik Opht., 1940, v. 17, pt. 5, p. 686.

The subject under discussion is the anatomy of the ciliary ganglion and nerves.

Ray K. Daily.

NEWS ITEMS

Edited by Dr. Ralph H. Miller
803 Carew Tower, Cincinnati

News items should reach the editor by the twelfth of the month

Deaths

Dr. Minot Flagg Davis, Cambridge, Massachusetts, died July 26, 1941, aged 78 years.

Dr. Claude William Walker, Iron Mountain, Michigan, died July 18, 1941, aged 65 years.

Dr. Alexander Green Touchstone, Meridian, Mississippi, died July 21, 1941, aged 54 years.

Dr. Walter Marion Haggett, Chicago, Illinois, died July 17, 1941, aged 83 years.

Dr. Frank Joseph Monaghan, Oneonta, New York, died July 26, 1941, aged 75 years.

Dr. Joseph Harris Ball, Cleveland, Ohio, died July 18, 1941, aged 68 years.

Dr. Gaven Crane Dyott, Eugene, Oregon, died July 15, 1941, aged 49 years.

Dr. Robert Leland Westover, Okmulgee, Oklahoma, died May 11, 1941, aged 68 years.

Dr. Philip John Lewert, Scranton, Pennsylvania, died August 27, 1941, aged 51 years.

Dr. Harry Perry Engle, Newton, Iowa, died August 5, 1941, aged 68 years.

Dr. Francis Bernard Chavasse, London, England, died in July, 1941, aged 53 years.

American colleagues share the distress of British ophthalmologists in learning of the sudden death following a motor accident in July, 1941, of Bernard Chavasse. According to the obituary notice in the September number of the British Journal of Ophthalmology, Dr. Chavasse graduated from Oxford with high honors in natural science and obtained all the scholarships of his year in his medical course, and at Liverpool went straight into the R.A.M.C. after qualification. He served with distinction during the war and won the Military Cross as the result of "gallant effort to save the life of his twin reported to be lying wounded in No Man's Land. For two nights he crawled about searching over a wide area, but in vain." While a medical officer he designed a splint for fractured femur that has been used in the present war. He is best known in this country for his rewriting "Worth's squint," and for his chapter on the development and pathology of binocular reactions in Ridley and Sorsby's book, "Modern trends in ophthalmology." He was secretary of the Medical Board of the Liverpool Eye and Ear Infirmary and devoted much of his time, at the expense of other pursuits, to the work on the Board, particularly in raising money for the equipment of the new Out-Patient Department and the establishment of an orthoptic clinic.

He is survived by his wife and three children.

Miscellaneous

The sixteenth annual Spring Graduate Course in Ophthalmology and Otolaryngology will be given at the Gill Memorial Eye, Ear, and Throat Hospital from April 6 to 11, 1942. Among the guest speakers will be: Drs. George E. Shambaugh, Jr., Chicago; George M. Coates, Philadelphia; Norton Canfield, New Haven; W. E. Grove, Milwaukee; Edwin N. Broyles, Baltimore; Warren Davis, Philadelphia; John R. Richardson, Boston; B. Y. Alvis, Saint Louis; Harvey E. Thorpe, Pittsburgh; Wendell L. Hughes, New York; Earl L. Burky, Baltimore; Edmund B. Spaeth, Philadelphia; and E. B. Dunphy, Boston.

The Eye Department of Emory University has established an aniseikonic clinic and an eye pathological laboratory through the generosity of Mr. L. F. Montgomery of Atlanta. The clinic has a full-time technician, and the work will be in charge of Drs. William T. Edwards, Jr., and Phinizy Calhoun, Jr., who have recently been added to the university staff.

The Harvard Medical School announces the following courses for graduates, beginning January 5, 1942: "Fundamentals of refraction" by Drs. Messenger and Sloane; "Clinicopathological ophthalmology" by Dr. Terry; "Ophthalmoscopy" by Drs. Regan and Sachs; "Ocular complications in general disease" by Dr. King; "External diseases of the eye" by Dr. Gundersen; "Biomicroscopy" by Dr. Beetham; "Principles of ophthalmic surgery" by Dr. Dunphy and staff; and "Recent advances in ophthalmology" by Dr. Terry and staff.

Societies

The Milwaukee Oto-Ophthalmic Society held its first regular meeting on Tuesday, October 14th. Dinner was followed by a clinical meeting consisting of presentation of cases. Dr. John Bellows, Chicago, spoke on "The nutritional aspects of cataract." A discussion on this paper was given by Dr. P. F. Swindle, Milwaukee. Dr. O. P. Schoofs lectured on "Maxillary sinusitis resulting from dental surgery."

Personals

A dinner in honor of Professor Emeritus John E. Weeks of the Department of Ophthalmology of New York University was held at the Union Club, New York. Eighty of his friends and students attended. The speakers program was as follows: "The development of

the laboratory side of ophthalmology" by Dr. Edgar Burchell; "Undergraduate ophthalmology in relation to the general medical course" by Dr. Currier McEwen, Dean, College of Medicine; "Graduate and postgraduate courses in ophthalmology" by Dr. John H. Dunnington, Professor of Ophthalmology, College of Physicians and Surgeons; "The relation of the special hospital to the university or general hospital" by Dr. David H. Webster, Executive surgeon, Manhattan Eye, Ear, Nose and Throat Hospital; "The American Board of Ophthalmology" by Dr. Conrad Berens, chairman, American Board of Ophthalmology; and "The chair of ophthalmology of the College of Medicine, New York University" by Dr. John E. Weeks, Professor Emeritus, New York University. Dr. Daniel B. Kirby, Professor of Ophthalmology, New York University, was toastmaster.

Dr. Weeks had received in 1923 the special degree of Doctor of Laws from New York University. The Eye Surgery Fund, Inc., a foundation for the training of eye surgeons and for the alleviation of blindness was dedicated to the memory of Dr. Webb W. Weeks. The poem "Spring and the blind children" has been dedicated to this Foundation by the author, Alfred Noyes. Two of the stanzas are as follows:

"They passed the primrose glistening in it dew,
With empty hands they drifted down the lane,
As though, for them, the Spring held nothing new;
And not one face was turned to look again.

Like tiny ghosts along their woodland aisle
They stole. They did not leap or dance or run;
Only at times without a word or smile
Their small blind faces lifted to the sun."

In the News Items of the October number it was erroneously stated that Dr. E. B. Dunphy of Boston would take part in the symposium on "Surgery of heterophoria and heterotropia" held at the thirty-first annual Clinical Congress of the American College of Surgeons. Dr. Moacyr E. Alvaro, who was the guest of honor of the College of Surgeons, spoke on the subject, "Recent advances in the surgery of heterophoria" in Dr. Dunphy's place.